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EXAMINATION OF THE CENTRAL NERVOUS SYSTEM.[1]

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When the Secretary suggested to me that a paper on a neurological subject would perhaps be of some interest to members, I must confess that I found myself in rather a quandary. Neurology has been the Cinderella of the family of medical sciences in Australia for many years; I shall suggest to you possible causes for this. First, neurological teaching has not occupied that prominent place in our curriculum which is allotted to it in the medical schools of other lands. Second, there is a widespread impression, in my opinion very ill-founded, that neurology provides a fascinating intellectual study for the neurologist, but merely empty words for the patient suffering from a nervous disease; to put it more bluntly, that the function of the neurologist is "to give the ill he cannot cure a name."

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on November 19, 1930.

I doubt whether gentlemen who hold this opinion are fully conversant with the advances which have been made in the therapy of nervous diseases during the past twenty years. Much there is still to do—but much is done already.

May I illustrate my point by a story.

A close friend of mine, a brilliant electrical engineer, now about fifty, left Australia for Canada some years ago. In January, 1929, he commenced to lose his memory; in March, 1929, he noticed a right-sided weakness, first in arm, then in leg; in May, 1929, he became aphasic. Early in June his physician in Vancouver diagnosed a tumour affecting the left frontal lobe. He was sent to Boston, where he arrived the last week in June. His tumour was removed under local anæsthesia by Dr. Harvey Cushing during the first week in July. Exactly six weeks after his operation that man dined with me in London, and looked, and said that he felt, perfectly well.

A third possible reason for the neglect of neurology may be that a neurological examination takes time—in certain cases a long time—in some cases, I suggest to you, a quite unnecessarily long time. Even the most conscientious of men, faced with a surgery still full at eight o'clock, may think it well nigh

impossible to investigate thoroughly a patient who complains of vague weakness in one leg. The temptation is always present to him to label the case as suffering from "one of those nervous diseases," and to salve his conscience by the reflection that, even if he did trouble to make a more exact diagnosis, he probably would not be able to do any more for the patient than he had done already.

Be the reasons what they may, the lack of interest in the more obscure problems of neurology is undoubted; hence my embarrassment in the choice of a neurological subject. It occurred to me, however, that when one wishes to interest a man in a problem, one first discusses with him the methods which can be used in investigating that problem, before one passes to the problem itself. If I am correct in that conclusion, then, to interest you in neurology, I should first discuss with you the technique of neurological investigation; and this I have decided to do.

As a basis for discussion I propose to set before you my own routine methods when I am confronted with a neurological case. I do not suggest that the majority of these methods are either original or new; on the contrary, many of them are tried and trusted veterans in the neurological world. Neither do I bring them forward as in any way comparable with the laws of the Medes and the Persians, a fixed, unalterable code; I change them as often as I learn better or quicker methods of eliciting the information I seek, and I am in hopes of changing them considerably for the better after tonight's discussion. I do, however, claim that a practised observer, following out the scheme which I present to you, can determine the presence or absence of organic nervous disease within twenty minutes, which is surely not an unreasonable amount of time to devote to making that matter clear. To be sure, the case of spinal or cerebral tumour may take rather longer for its elucidation, but what complicated medical case does not? The value of having an adequate routine method of examination is not always sufficiently appreciated. If one is tired—if one is very busy—how easy it is to forget the very thing needed to establish a diagnosis in that particular case. Is it not very true that far more diagnoses are missed through not looking than through not knowing? A good technique, mind you, is not acquired without some labour and much diligent practice; but when you diagnose a condition which has been missed time and time again and cure the patient, then you have your reward. The cricketer relies upon a technique so often practised as to be almost a part of himself when he is faced with a difficult good length ball on the off stump; the tennis player can almost without thinking unwind that backhand drive down the line, which is such a present help in trouble; the gunner can handle his complicated mechanism under the most trying circumstances because he has done it so often before; and the neurologist must without having to think unduly hard be able to collect the information he requires with speed, with accuracy and with completeness. To those who say that in advocating a somewhat stereotyped method I am discouraging original thought, I would reply that most of us are capable of only a limited amount of original thinking in the day, and it is surely wasteful to spend that precious modicum on what should be mere routine work.

One word of apology—if I seem at times to be describing trivial and elementary details, with which you are all acquainted, I ask of you to bear with me patiently; it is just in these simple points of technique that mistakes are made time and time again. How often have you seen plantar reflexes which have in some mysterious manner changed the nature of their response in the bare half-hour between the visits of two doctors? I am going to run the risk of boring you, and discuss these and similar matters in detail; my paper, like the lady's dress, will have to be long enough to cover the subject; it is my hope that it, too, may be short enough to be interesting.

The History.

One starts with the taking of an accurate history. Time will not permit me, nor do I think it necessary to discuss this matter with you in detail. On certain subjects you will on no account omit to obtain information; in the past history remember the possible influence of syphilis, of heart disease, of alcohol, and of occupation; in the family history learn all you can about any cases of nervous disease in relatives; and, coming nearer home, in the history of the present illness try to get as clear a picture as possible of the order of occurrence of the different symptoms which make up the present clinical picture.

While you take this history you will get some sort of impression as to the mental condition and intellectual powers of the patient. This may be of diagnostic assistance; many organic nervous diseases are accompanied by a characteristic change in mood. It will also give you a good idea of how much cooperation you can expect from the patient in the subsequent objective examination; and you will adapt your methods and interpret your results accordingly.

In discussing the actual methods used in a routine examination of the nervous system, I shall first speak of those essential tests which I carry out in every case, and which come under the heading "Routine Methods" in the summary which you have in your hands; I shall then discuss additional methods, of value in individual cases; and I shall finally mention certain subsidiary methods, of great diagnostic worth, but which do not form part of the physical examination, properly so-called.

The Special Senses.

Sight.

I begin with the examination of the special senses, taking sight first. The simple question, "Do you see so-and-so (pointing to some object) equally well with either eye (covering each in turn)?" often suffices. Where defective vision is suspected, I use the ordinary Snellen type card.

I then carry out a rough examination of the visual fields with my fingers, to establish the presence or absence of a hemianopia, of which the patient himself may be completely unconscious. If a field defect

is suspected, then more exact perimetry will be required; for clinical work I use a Schwarz hand perimeter. I admit that this instrument is not so accurate as the more elaborate installations of my ophthalmological brethren; but in all except very small field defects it gives sufficiently reliable information. With this instrument one can, with a little practice, quite easily delimit such a field as this, where you will see the typical quadrantic defect of a pituitary tumour. If you have not a perimeter of any sort, a hatpin and a small disc of white paper are a great deal better than nothing. Field defects are so common and so important that every neurological physician must acquire a reasonable degree of proficiency in their detection. Lest it be thought that in saying this I am trespassing upon the preserves of the ophthalmologists, I would remind those gentlemen that perimetry owes its development at least as much to the neurologists of Queen Square as to the ophthalmologists of Moorfields; it is a useful weapon in both our armouries.

I then examine the *fundus oculi*, noting particularly the state of the disc, the condition of the retinal vessels and of the retina itself, and the presence and nature of any hæmorrhages or exudates. With a little practice most fundi can be examined fairly satisfactorily without dilatation of the pupil. This is especially desirable where any question of pupillary abnormality may arise. I have known the effects of an instillation of homatropine to persist as long as ten days after the pupil was dilated, even though eserine was used; pity the lot of the unfortunate consultant, trying to ascertain the condition of the third nerve in such a case. If you must dilate the pupil, examine the third, fourth and fifth nerves thoroughly first.

Hearing.

The sense of hearing comes next. For rough testing it is generally sufficient to ascertain the distance from each ear at which a watch can be heard; the distances noted for each ear are compared with each other and with the normal. Where there is any diminution of auditory acuity, you will differentiate middle-ear deafness from nerve deafness by the Rinne and Weber tests, the details of which are well known to you, and no examination of the ear is complete without an inspection of the *membrana tympani*.

Smell.

I do not investigate the sense of smell as a routine in every patient; where for any reason this seems indicated, I use lavender and cloves, and note the distance from each nostril at which each odour is perceived. Unilateral anosmia may be of the greatest value in diagnosis, and may finally determine the localization of that not uncommon and operable cerebral tumour, the meningioma of the olfactory groove; but do not diagnose nervous anosmia in a patient with a cold in the head.

Taste.

There is still considerable discussion as to the exact pathway taken by the nerve fibres subserving taste. It is a very difficult sensation to test; rarely, if one excepts an occasional facial palsy, does it give information not obtainable by other and easier means; I do not include any tests of taste in my routine examination.

The Cranial Nerves.

III, IV, VI. Next come the cranial nerves. The third, fourth and sixth nerves form a group and can conveniently be examined together. In examining the pupils I observe their size, shape, reaction to light and reaction to accommodation. It is easy enough to mention those examinations, easy enough too to carry them out; but for all that, there are many mistakes made in pupillary examination, for want of a few simple precautions.

1. Do not examine the size of the pupils in too strong a light; pupillary contraction produced by this light may mask an inequality in size, which is easy to determine when the eyes are semi-dilated.

2. Take particular care over the shape of the pupils; apart from congenital coloboma and from the results of iridectomy, any irregularity of outline is almost certainly syphilitic.

3. In taking out the reaction of the pupils to light, remember to observe their contraction both to direct and to consensual stimuli. It is better to use daylight for this reaction, but if for any reason this is impossible, be careful to have the source of light out of the patient's visual field; a contraction of the pupil when a torch is flashed in the patient's face may be merely the contraction of accommodation produced by the eyes being focused on the light. I am convinced that many Argyll-Robertson pupils are missed by neglecting this simple precaution. Remember that a sluggish reaction to light may be almost as important as a complete absence of reaction.

4. Reaction to accommodation is most simply tested by directing the patient to look out of the window (not at the window), and then at the examiner's finger.

If the patient then follows the examiner's finger with his eyes—you may have to fix the head with your hands—while the finger is moved laterally, moved vertically and finally circumducted, any defect in external ocular movement will be made plain.

While doing this, one can inquire as to the presence of any diplopia. It is not always easy to identify the particular muscle which is paresed when any external ocular movement is defective; charting the apparent positions of true and false images where there is a diplopia may be of considerable help. The patient wears a pair of plain tinted glasses, the right glass green, the left red. I then hold a lighted candle vertically in front of him, and place it successively in nine different fields in relation to this direction of gaze: upper right, upper central, upper left, right, central, left, lower right, lower central, lower left. On each occasion the patient tells me whether there is diplopia or not, and, if there is, what are the relative positions of the green and red candles. I chart these findings as shown in Figure I; this is a case of simple left lateral rectus paralysis.

It is easier to draw the chart from the point of view of the patient rather than of the observer; and it is

useful to remember, in interpreting such a chart, that the false image is displaced in the direction of traction of the paralysed muscle, that is, in this case, directly to the left.

Ptosis you will have noted before this.

Nystagmus you will also have detected. Where you have seen a nystagmus it is worth ascertaining:

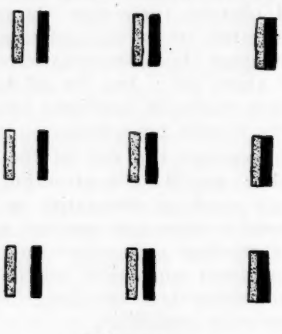


FIGURE I.

(i) Whether it is spontaneous or not, (ii) whether it is fine or coarse, (iii) its direction, lateral, vertical or rotatory, (iv) the direction of the slow component, (v) the direction of gaze in which it is most marked. In order to determine whether a spontaneous nystagmus has anything to do with the visual apparatus, or whether it is of purely labyrinthine origin, I use a turning umbrella. If I spin this round the head of a normal patient he will develop a nystagmus; if I spin it round the head of a patient with a spontaneous nystagmus of mid-brain origin, some alteration in the rate, quality or direction of the nystagmus will be produced; but if I spin it round the head of a patient with nystagmus of purely labyrinthine origin, the nystagmus will not be altered in any way whatsoever.

V. The fifth nerve has both motor and sensory branches; the former supply the muscles of mastication. "Open your mouth!" "Shut it tightly!" is sufficient to demonstrate to the examiner's fingers whether the temporal muscles are working equally and effectively; "Open again!" "Shut again!", and the masseters are similarly examined.

More important is the examination of the sensory branches. The corneal reflex is a most delicate indicator of sensory integrity, but the usual method of eliciting this reflex, touching the cornea with cotton wool, is apt to be unpleasant for the patient; moreover, you may get a false response, a blinking brought about by the sight rather than by the touch of the cotton wool. Both these objections are met by the very simple method of directing a gentle current of air from the examiner's mouth against the cornea, and observing the blink thus produced.

I go quickly with a wisp of cotton wool over the areas supplied by the three branches of the sensory root; this is my routine method of investigating the sensation of light touch. Here, as elsewhere in the body, I prefer to instruct the patient, whose eyes are shut, to say "Yes" each time he feels anything touch him, rather than to keep on asking: "Do you feel this?" If any anæsthesia is present, the patient is not made aware of it, as I keep touching areas where I know perception is unimpaired sufficiently often to keep him interested, and to prevent him from getting the impression that he is being touched and not feeling it; many an hysterical anæsthesia has been created or made worse by neglect of such a simple precaution.

I then go over the same area again with a pin. Instead of saying: "Do you feel this?" or "Is this sharp?", I prefer to say: "I am going to touch you with a pin, now with the sharp end and now with the blunt. Please say sharp or blunt each time you are touched."

If any loss or blunting of sensation is present, I try to define its limits accurately; the characteristic "butterfly" analgesia of tabes is, of course, well known to you. (See Figure II.)

It is not necessary to investigate other forms of sensation in this area in a routine examination.

VII. In investigating the seventh or facial nerve, I observe, first, the state of nutrition of the muscles supplied by that nerve, and, second, whether any contractures are present. In the early state of a Bell's palsy, there is usually little difficulty in making the diagnosis of right- or left-sided facial paralysis, as the case may be, even when the face is at rest; the smoothing out of the frontal furrow, the flattening of the naso-labial fold, the drooping of the mouth present a characteristic picture; but when the condition is one of long standing, I have known men of considerable experience diagnose the paralysis on the wrong side, being led to that belief by the

FIGURE II.

effect of secondary contracture in the paralysed cheek muscles, which bring it about that the angle of the mouth on the paralysed side is actually higher than on the sound side. A little care in investigating facial movements will readily obviate such a mistake; only the spot diagnostician is likely to fall into error.

The investigation of these facial movements is a task of a few seconds only; it takes far longer to describe than to perform. First notice whether that patient blinks equally and normally with either eye; inequality in blinking may be the first sign of a facial nerve lesion. Then give him the following commands: "Wrinkle your forehead!", or better "Look right up!". You will remember that when the lesion causing a facial paralysis is situated below the facial nucleus, the patient is unable to wrinkle his forehead on the affected side; when the lesion is above the nucleus, as, for instance, in a capsular thrombosis, the forehead muscles escape, though the muscles of the lower part of the face may be completely paralysed. "Close your eyes tightly, so that I cannot open them!" Although both eyes may appear to be equally well-closed, an attempt to open them with the fingers will often reveal considerable difference in the muscular power on the two sides.

"Blow out your cheeks!" requires no comment.

"Show me your teeth!" That is the classical form of the command, but I find it better to say, "Wrinkle up your lip like this!" I am thus saved from the possibility of getting as a reply either "Sorry, doctor, I haven't any," or "It's not worth looking, doctor, they are all false." If I then explain that I am not interested so much in their teeth as in the action of their facial muscles, I am always liable to the even more disconcerting reply, "Oh, I am sorry, doctor, why didn't you say that in the first place?" On the whole I prefer to ask my patients to: "Wrinkle up the lip like this!"

A request to whistle may remove any doubts still lingering in your mind about a possible mild weakness in the muscles round the mouth.

VIII. The two components of the acoustic or eighth cranial nerve are investigated independently of one another. The cochlear nerve we have already discussed in dealing with the testing of hearing, and it requires no further mention. The vestibular nerve I do not investigate in the course of a routine examination, unless there is some special indication, as, for example, the presence of dizziness, tinnitus or deafness, that this should be done. If any of these symptoms are present, however, and the question of the intactness of the vestibular nerve and the labyrinth arises, I make use of Bárány's caloric tests. The details of these tests are somewhat lengthy, and I do not propose to discuss them here. Suffice it to say, that Bárány has demonstrated that where the vestibular system is intact, the introduction of cold water or cold air into the external ear on one side will produce nystagmus of a certain type and in a certain direction, depending on the position of the patient's head at the moment of testing; hot water or hot air will, in general, produce an exactly opposite effect. The patient will, moreover, under the influence of this stimulation, tend to bend his body in a certain direction, again depending upon the position of his head, and may demonstrate the so-called "past pointing"; that is to say, when he is asked to move his hand directly up and down, it will tend to deviate to one side or the other as the case may be.

The failure of the caloric stimulation to produce these phenomena is an indication of some abnormality in the vestibular system; with the precise interpretation of results obtained by these methods I will not here concern myself.

Placing the patient in a revolving chair and spinning him round a certain number of times is an additional method of investigating the vestibular apparatus; one observes the nystagmus and the disturbance in equilibrium thus produced, and interprets them according to the rules laid down by Bárány. I have no great liking for this method, either as patient or as investigator; it may produce most distressing vertigo and vomiting; and it is seldom that one gains any information of diagnostic value beyond that obtained from the caloric tests.

IX, X. Observation of the movements of the soft palate on saying "a--a--ah" will generally indicate whether any defect is present in the vago-glosso-pharyngeal system. Confirmatory evidence of a suspected loss may be obtained by very gently touching the posterior pharyngeal wall with a probe—the resultant pharyngeal reflex is an indication of the integrity of the reflex arc. You will remember, too, that the vagus supplies the muscles activating the vocal cords, and it is a simple matter to inspect the movement of the cords on respiration and phonation when a possible vagus paralysis is in question.

XI. The eleventh nerve supplies, among other structures, the sterno-mastoid muscles. I look for any wasting in these muscles, and test their power by getting the patient to move his jaw to one side and then the other against the resistance of my hand.

XII. Ask the patient to protrude his tongue. After observing any tremor, fibrillation or wasting, you will particularly note the direction of protrusion in relation to the mid-line of the upper jaw (not, be it remembered, of the mouth; if this latter is your guide, a coexistent facial palsy will often deceive you). A tongue organically paralysed on one side protrudes to that side, but when the tongue is withdrawn completely into the mouth it tends to pass across to the other side; but it is interesting to note that the hysterical patient with a deviated tongue, not knowing his anatomy, still keeps his tongue on the same side when he retracts it into the mouth again.

So much for the cranial nerves. Although I have spent a considerable time on discussing methods of investigating them, the actual performance of the methods is usually a very much shorter affair, involving a few minutes only.

The Motor System.

Next comes the examination of the motor system. Concerning the best methods of investigating the cranial nerves there is considerable unanimity, and the methods of one neurologist may differ little from those of half a dozen of his *confrères*. When one comes to the motor system, the situation is quite otherwise, and no two men seem to set about the task in quite the same way. A man who has been trained under several teachers, whose methods differ radically from one another, often has a very jumbled

view of the whole affair; his examination of the motor system is liable to consist of the rule-of-thumb performance of a number of tests, without his having any very clear idea at the back of his mind what particular functions he is actually testing. The best test is that which gives the greatest amount of definite information in the least time, and each test employed must be judged accordingly to that standard. I shall put before you my own selection from the many methods available; I hope that you will help me to make a better one.

It is not possible to devise tests for the investigation of each separate motor function individually; a certain amount of overlap is unavoidable; I have tried to reduce this overlap to a minimum.

I investigate the motor system under six heads: (i) General inspection, (ii) posture maintenance, (iii) tonus, (iv) rate of initiation of movements, (v) strength, (vi) coordination.

General Inspection.

In the general inspection I note particularly the state of nutrition of the muscles, and if there is any wasting present, I determine its exact distribution, and observe whether any involuntary movements are present, and if so, their distribution and their character.

Posture Maintenance.

To determine how the patient maintains posture, I ask him to stretch his arms out horizontally in front of him, palms down, fingers separated, and when he has taken up that attitude, to maintain it. I look to see whether the voluntary exertion has produced a tremor which was not present when the arm was at rest; it is possible, on the other hand, that a previously observed tremor will now vanish. The former phenomenon may indicate an early disseminated sclerosis; the latter is not unknown in mild cases of Parkinsonism. I look to see whether either arm droops or in any way changes posture spontaneously. I then tell the patient to try not to let me move his arms, and I displace them upwards, downwards and sidewards with my hands, noting the amount of resistance met with on either side, and the quickness and accuracy with which the limbs return to their previous posture.

Similar observations may be carried out on the legs; the patient lies on his back and lifts his legs a little from the bed, knees straight and heels apart. Fatigue comes on more quickly in legs than in arms, and posture control is never so accurate in the lower as in the upper extremity; nevertheless, the information gained by investigating posture maintenance in the legs may be of value.

Tonus.

Passive manipulation of the resting limbs is the easiest method of investigating tonus. I classify my findings as (i) diminished tonus, (ii) normal tonus, (iii) increased tonus, which latter is synonymous with rigidity. Rigidity itself can readily be differentiated into three types; the nature of the rigidity may be of considerable value in helping to determine the site of the lesion.

In the "clasp knife" rigidity, produced by a pyramidal lesion, it may take a considerable effort on the part of the examiner to initiate flexion at a joint, but once begun the movement is carried through smoothly and easily.

In the "cog wheel" type of extrapyramidal rigidity, the resistance waxes and wanes throughout the passive movement, which proceeds in a series of small jerks. The rigidity of Parkinsonism is of this type.

Hysterical rigidity provides an even and uniform resistance throughout the movement. To produce this rigidity the patient must be concentrating on the limb under examination; hence if, after examining one arm, you leave one of your hands carelessly resting on that arm, and proceed to the examination of the other, you will feel the excessive tonus gradually fade out of the muscles until it has quite disappeared. Now if you jerk the limb up suddenly, you may almost without effort carry out a movement which a few minutes earlier taxed all your strength; but your patient will not necessarily like you any the better for demonstrating the purely functional nature of his ailment.

Rate of Initiation of Movements.

You will notice the rate at which movements are initiated when you are testing the strength of the individual movements; of particular interest is a difference in rate between the two sides of the body.

Strength.

It is neither necessary nor possible when testing strength of movement to investigate the action of every muscle or group of muscles in the body. The movements which I shall mention to you involve almost all the spinal segments for their execution; if you want to investigate any particular segment more closely, it is not very difficult to devise suitable methods. It is understood that in each case the movements are to be carried out against a resistance provided by the examiner.

In the arms, I make use of flexion and extension at the elbow, pronation and supination, flexion and extension at the wrist, the finger grip.

In the trunk, I ask the patient, who is recumbent, to flex his chin on his chest and then to sit up, or attempt to sit up; I note any palpable difference in the contraction of the different groups of abdominal muscles, and any variation in the position of the umbilicus indicating some abnormality in muscular contraction.

In the legs, the patient being recumbent, raising and lowering the knee and dorsiflexion and plantar flexion at the ankle generally suffice.

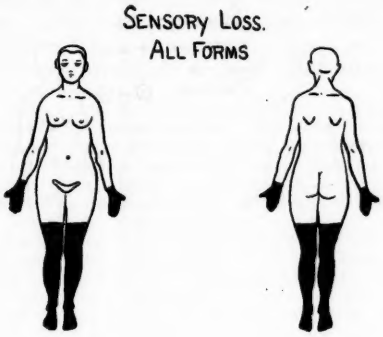
Coordination.

In investigating coordination, I employ as a routine three tests only—one can devise dozens more just as effective—almost any complex movement can be made the subject of a test.

I ask the patient to touch alternately his nose and my outstretched finger, his eyes being first open, then shut.

I ask the patient to extend his upper arms horizontally in front of him, and then to rotate the forearms, which are held vertical, as rapidly to and fro as possible.

I ask the patient, who is lying on his back, to place each heel in turn on the opposite knee, and then to bring that heel down along the line of, but just clear of the anterior edge of the tibia till it reaches the ankle.



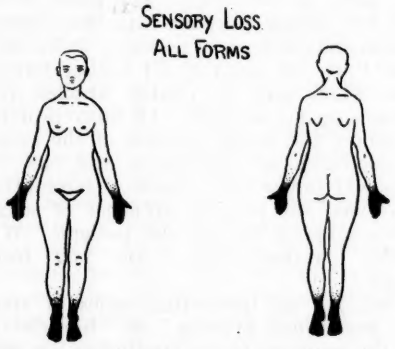
Hysteria
Figure III.

Any incoordination which does not demonstrate itself in the performance of these three tests is not likely to be of any considerable clinical significance.

That concludes the examination of the motor system; once again the time required to describe the methods used is far greater than that needed for their performance.

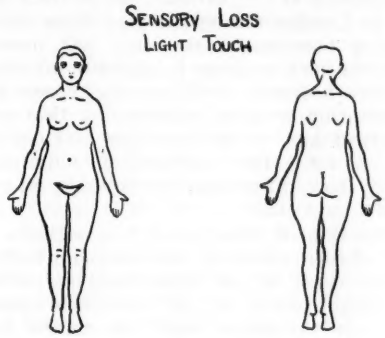
The Sensory System.

Coming to the sensory system, one is again on well-trodden ground and the methods of examination are fairly stereotyped. Yet nowhere in neurology is there more scope for the utilization of skill and



Peripheral Neuritis
Figure IV.

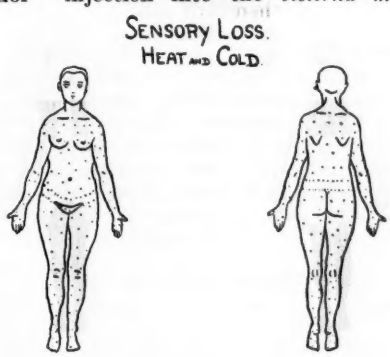
experience than in the exact mapping out of areas of sensory loss. It is a task which calls for a knowledge of the art no less than of the science of medicine; and it is indeed an intellectual treat to have the privilege of watching a gifted exponent of that art at work. The areas of anæsthesia or analgesia seem to delimit themselves before you in the course of a very few minutes; you may yourself have spent as many weary hours on the same patient, without getting anything like such satisfactory or clear-cut



Tabes.
Figure V.

results. Indeed, the very fact that you have spent hours on the job may in itself be one of the factors contributing to your failure; the patient's attention rapidly wanders, and it is often better practice to stop after ten minutes and start again on another occasion, than to attempt to obtain all your information at one sitting. The effort involved in acquiring some skill in this field of investigation will be very handsomely repaid. If you localize a spinal tumour accurately, and as a direct result a patient walks and runs, who had given up all hope of walking or running again, is not that sufficient reward?

And how, if not by an accurate sensory examination, are you going to localize such a tumour? "Lipiodol" injection into the *cisterna magna* is



Tabes.
Figure VI.

at best a subsidiary method; the injection itself is one only for practised hands, and, moreover, false localization has been reported—the "Lipiodol" has been arrested elsewhere than at the site of the tumour.

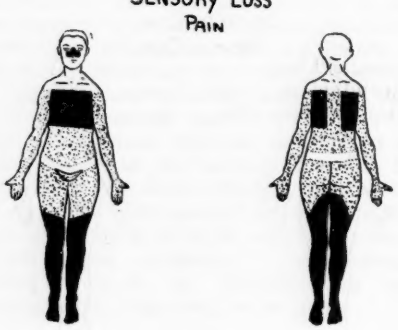
Further, no surgeon will thank you for making an already difficult task more difficult by obscuring his operative field with a viscid brownish mess.

I do not suppose that "Lipiodol" has been injected into one-third of all the suspected cases of spinal tumour at the National Hospital for Nervous Diseases in London during the past three years, and the number becomes yearly less; yet nowhere in the world are such tumours localized more accurately than in that hospital. It is a daily reproach of the older generation to us of a later day that we have never learned how to use our eyes and our hands; I know of no better method of acquiring that proficiency than in learning how to make an accurate sensory examination.

The sensation of light touch I investigate with a fine wisp of cotton wool in the manner which I have already described to you when speaking of the fifth nerve. Light touch is, as you will remember, conducted up the spinal cord both in the posterior and in the lateral columns of white matter, and it is rare that a spinal lesion is so gross as to interrupt completely both of these pathways; hence it is rare for light touch to be completely lost where the lesion is in the spinal cord.

Pain is investigated with the blunt and the sharp ends of a pin; that, too, we have already discussed. Perception of heat and cold is readily tested by the use of a pair of test tubes, the one containing hot, the other cold water. Pain, heat and cold are all conveyed in the lateral columns of white matter in the cord; hence the areas of diminution or loss of all these forms of sensation may closely resemble one another in cord lesions.

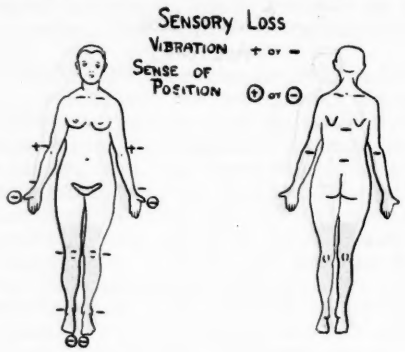
It is a good general rule, in investigating all these forms of sensation, to pass from the anæsthetic to the normal area, rather than *vice versa;* it is much easier for a patient to determine when he first



Tabes
Figure VII.

perceives a sensation, rather than when that sensation disappears.

I chart my results on simple diagrams made with the aid of rubber stamps. When the area of anæsthesia is the same for all forms of sensory stimulation, as in a case of hysteria (Figure III), one pair of diagrams will suffice; similarly in a case of peripheral neuritis (Figure IV) there is no need of more than one pair. In most cases, however, I prefer to plot each form of sensation separately; thus in a



Tabes
Figure VIII.

tabetic under my observation, the loss to light touch (Figure V) was nil; the loss to heat and cold was as you see it in Figure VI; the loss to pain (Figure VII) was far more extensive. In passing, I would draw your attention to one or two features of particular interest in the tabetic distribution of analgesia: (i) The sharp line of demarcation between the fourth cervical area, where the patient feels a pin prick perfectly, and the second thoracic, where he feels it not at all; (ii) the "cuirass" of absolute analgesia; (iii) the frequent complete escape of the twelfth thoracic segment. All these features are present in this particular case.

The posterior column of white matter is the pathway for the proprioceptive sensations from the different parts of the body. The most convenient of these for testing purposes are the appreciation of vibration and the sense of position. In investigating vibration I use an ordinary C1 tuning fork with a flat base which can be readily applied to bony prominences or flat surfaces. Of bony prominences, the olecranon, the styloid process of the radius, the lateral epicondyle of the femur and the external malleolus will suffice for a routine examination. In order to avoid the possible influence of suggestion, it is always better to ask the patient "What do you feel?" rather than "Do you feel that vibrating?"

You will get an interesting series of similes in reply. "Something buzzing" or "like electricity," or when the resources of the vocabulary are not equal to the occasion, "I don't quite know, doctor, but it feels funny," can all be reasonably accepted as positive answers.

I am inclined to doubt the value of any information received by applying the fork to the spines of the vertebræ; some observers claim that this method provides valuable help in localizing a spinal cord lesion. I have seen several cases of spinal tumour in which vibration was perceived when the fork was applied at least half a dozen segments below the

level of the tumour, as subsequently ascertained by operation; I suggest that in these cases the vibration is transmitted directly up the bony column until it reaches an intact segment, by which it is appreciated and transmitted to consciousness.

There is, however, another use of the tuning fork, which I owe to my teacher, Dr. Gordon Holmes; the value of this particular method was impressed on me very strikingly by one interesting case.

The history and the nature of the cerebro-spinal fluid were characteristic of spinal tumour. The areas of loss to pain, heat and cold were extremely vague, very difficult to define and of no localizing value—light touch was, as it generally is in such cases, almost unimpaired. "Lipiodol" had been injected and had passed through to the bottom of the vertebral canal. Finally, when we had almost despaired of finding a level, Dr. Holmes suggested that applying the tuning fork to successive areas of pinched up skin might be of some help; I did this, applying the fork to each successive segmental area from the lower lumbar region upwards, and trying as far as possible to keep uniform the amount of skin pinched up, the pressure with which the fork was applied, and the amplitude of the fork's vibrations. The result was a clean cut level between the eighth and ninth dorsal segments; operation revealed an extramedullary tumour pressing on the posterior aspect of the cord, interfering chiefly with the posterior columns and only very slightly with those tracts lying more anteriorly; there was not sufficient obstruction of the subarachnoid space to arrest the "Lipiodol."

In the investigation of the sense of position a routine examination will be confined to the thumb and the big toe. These are in turn moved backwards and forwards, and the patient is asked to say when and in which direction a movement has taken place. Be careful to hold the thumb or toe lightly, forefinger on one side, thumb on the other; you thus avoid pressure on the dorsal or vertebral surfaces of the digit, which may give the patient a clue to the direction of the movement.

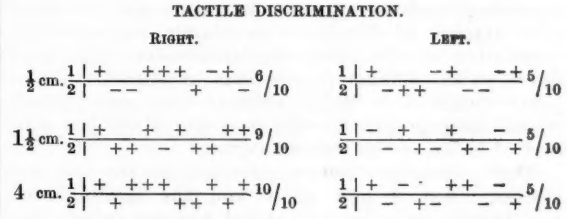
A second method of investigating sense of position is to place one of the patient's limbs in a certain position in space, and to ask him to place the other limb in a corresponding position; the eyes are, of course, shut as in all forms of sensory examination. It will be obvious to you that the accurate performance of this test involves not only a knowledge of the position of the limbs, but also a correctly functioning motor system.

Results of investigation of posterior column sensation may be charted thus; the posterior columns are, of course, severely affected in tabes (Figure VIII).

The sensory cortex is concerned with the appreciation of fine differences of sensation, and with the correlation of a number of individual sensory impressions not only with each other but also with the recollection of similar impressions received on previous occasions. As you will readily appreciate, certain aspects of cortical function lend themselves more easily than others to objective demonstration; I employ localization of touch and tactile discrimination as my routine methods, and stereognosis to confirm my findings.

Localization of touch is readily combined with the investigation of light touch and of pain, the patient being asked to place his finger on the exact spot touched or pinched; it is worth remembering in carrying out this investigation, that localization in the upper extremity is far more precise than in the lower, and that an error of two to three centimetres or even a little more in the latter may not be of any significance whatsoever.

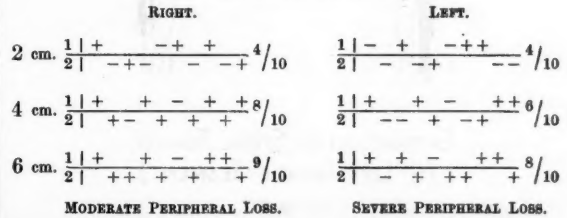
Tactile discrimination is the ability to distinguish between the sensation produced by the application to the skin of one point or of two points; in each case one applies the point or points with a uniform pressure. The palms and soles are the areas most suitable for testing. I use a pair of compasses with blunt downward deviated points; on one arm is a scale to indicate the distance between the points; any metal worker can make a similar pair in an hour or two. I commence on the palms with the points one centimetre apart and quickly touch various spots, now with one point, now with two; ten such touches can be done within a minute. I record my findings as shown in Figure IX. I then increase the distance between the two points say to two centimetres and repeat the procedure; after that I choose whatever distance seems appropriate to the case under investigation. The threshold in normal palms, that is to say, the minimum width between the two points at which the patient gives at least eight out of ten correct answers, is about one centimetre or perhaps a little more. The method of ascertaining this threshold will be obvious to you.



RIGHT FRONTO-PARIETAL GLIOMA.

FIGURE IX.

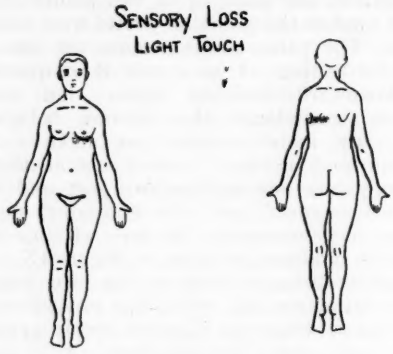
In certain cases of peripheral nerve disease this threshold may be raised, owing to the interference with conduction; nevertheless, by increasing the distance between the compass joints, a threshold will ultimately be reached (Figure X). Where the lesion is in the cortex, however, the distance between the points is of no significance; you are just as likely to get a correct answer at one centimetre as at six centimetres (Figure IX).



PERIPHERAL NEURITIS (MILD).

FIGURE X.

Stereognosis consists in the identification, without the use of the eyes, of objects placed in the hand. A key, a coin, a penknife are suitable and always available. Stereognosis is a highly complex faculty; it implies accurate localization of touch, accurate

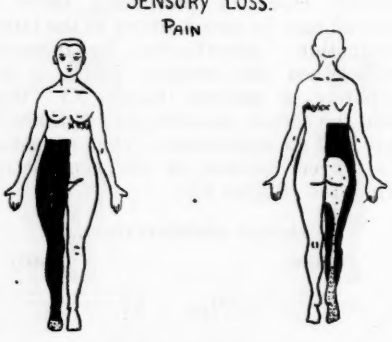


Extramedullary Spinal Tumour
(6th Left Thoracic Segment)

Figure XI.

appreciation of the variation in nature and intensity of a number of simultaneous stimuli, and accurate correlation of the composite impression thus produced with similar impressions produced in the past; hence it is highly unlikely that any patient whose stereognostic faculty is unimpaired has any sort of lesion of the sensory cortex.

That concludes the examination of the sensory system. May I now show you the diagrams of my findings in a case of spinal tumour, where the sensory loss was of the utmost localizing value (Figures XI to XIV). By way of contrast such a finding as this is possible in one condition only (Figure XV).

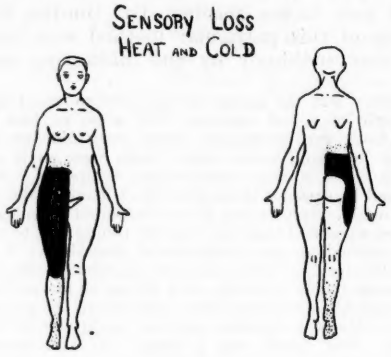


Extramedullary Spinal Tumour
(6th Left Thoracic Segment)

Figure XII.

Reflexes.

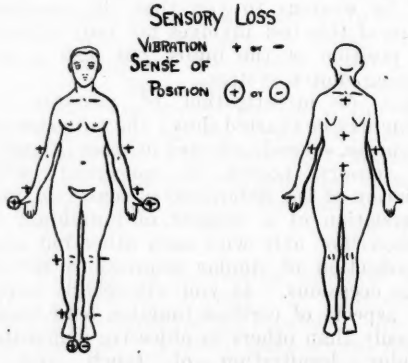
Of reflexes it may truly be said that their name is legion; everybody who writes a neurological book feels it his bounden duty to add a new one to the list; one feels tempted to paraphrase the old preacher and say that of making many reflexes there is no end, and that much study of reflexes is a weariness of the flesh.



Extramedullary Spinal Tumour
(6th Left Thoracic Segment)

Figure XIII.

Choose from the list those reflexes which are easy to elicit; scatter your selection among the different segments, first, so that no important area of the cord is not represented, and, second, so that there is no unnecessary duplication of work. Will you be good enough to excuse me if I seem, in describing reflexes, to deal with details apparently too trivial for your consideration. As a consultant one sees so many reflexes incorrectly elicited, that a little attention directed toward the acquirement of an exact technique would, I think, not be misplaced. The interpretation



Extramedullary Spinal Tumour.
(6th Left Thoracic Segment)

Figure XIV.

of a neurological condition may be difficult enough in the most favourable circumstances; with incorrect data it becomes quite a hopeless affair.

The actual instrument used for percussion is not without some importance. Personally I make use of a home-made affair; a rubber pessary fitted round a metal disc into the centre of which a cane handle is secured. This has the advantage that it

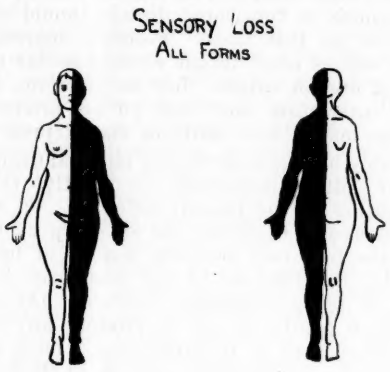


Figure XV.

stimulates a large area, and hence the receptor field, which may be small, is not so likely to be missed as it is with a small instrument; moreover, less force is required, and one is less likely to cause the patient pain than one is with these vicious little brutes.

A reflex may be absent, diminished, normal, increased, or its character may be altered; these are the symbols I use, and which I record on diagrams similar to those you have already seen (Figures XVI and XVII). I examine the reflexes in cranio-caudal sequence.

REFLEXES.

− Absent.
+ − Diminished.
+ Normal.
+ + Increased.
+ + + Greatly Increased.

Figure XVI.

The supinator jerk (C. 5, 6) is elicited by holding the patient's forearm loosely in a horizontal, half-supinated position, and striking the radius just proximal to the styloid process; the supinator longus and the other flexors of the elbow will be seen to contract.

The biceps jerk (C. 5, 6) may supplement information derived from the supinator jerk. Standing in front of the patient, the examiner supports the latter's forearm on his own left forearm and places his left thumb on the biceps tendon at the bend of the elbow; a gentle blow on this thumb will produce a reflex contraction of the biceps muscle.

The triceps jerk (C. 7, 8, T. 1) is not quite so easy. The patient's arm is held horizontal with the forearm flexed to a right angle; it is absolutely essential for the muscles of the arm to be completely relaxed. One then localizes exactly the tendon of the triceps about 2·5 centimetres (one inch) proximal to its insertion in the olecranon, and strikes this tendon fairly in the middle. It is very easy to strike to one or other side of the tendon, and report the reflex as absent; on the other hand, the mere mechanical stretching of the triceps produced by the tension in the tendon is sometimes mistaken for a positive response. It is perhaps not generally known that the triceps jerk may disappear in tabes before either knee or ankle jerks are affected; hence the importance of a correct technique.

There are three reflexes involving the lower cervical segments, which I do not include in a routine examination, but which are often of value in determining the exact nature of a paresis affecting the upper extremity:

1. To elicit Leri's forearm sign, I stand in front of the patient whose arm, slightly bent at the elbow, is held out, palm upwards, towards me. I clasp the outstretched fingers and forcibly flex them on the palm, continuing the movement until the wrist also is forcibly flexed. In the normal individual and also in the patient with a functional paresis the elbow will then be involuntarily flexed, and the forearm brought to a standstill against the upper arm; in an organic paresis, on the other hand, this flexion at the elbow will not take place.

2. Mayer's thumb reflex is elicited by forcibly flexing the middle finger of the hand to be examined at the metacarpo-phalangeal joint; under normal circumstances the thumb will immediately become opposed at the metacarpo-phalangeal joint, and extended at the interphalangeal joint. This reflex is characteristically absent in an upper motor neurone paresis of the arm.

3. The grasp reflex is a phenomenon of comparatively recent discovery; it was first reported by Adie and Critchley in 1926, and has since been found by a number of other observers. The reflex occurs only in connexion with lesions of the frontal lobe; the

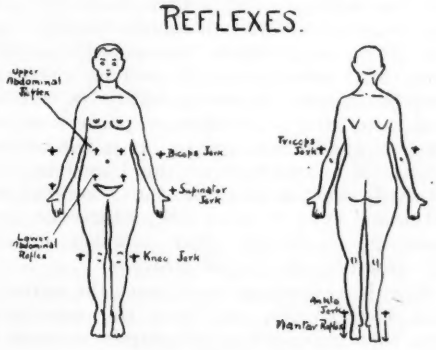


Figure XVII.

nature of the lesion appears to be of no significance. Of the three cases which have come under my observation, two were cases of frontal tumour, the third one of frontal abscess. The hand on the side opposite the lesion may be completely paralysed for

voluntary movement; yet if one draws a pencil or similar object across the fingers of that hand they will coil themselves round it and grasp it firmly; the harder one pulls, the tighter the grip becomes, until finally one can pull the patient half out of bed and even then his fingers will not let go; needless to say, the grasp is entirely beyond the patient's control.

As a rule, little technical skill is required for eliciting the abdominal reflexes (upper: T. 9, 10; lower: T. 11, 12); four diagonal strokes across the four quadrants of the abdomen, and the thing is done. When one is dealing with fat, flabby pendulous abdomens or, it is interesting to note, with an "acute abdomen," it may be very difficult to obtain the reflexes; but then the absence will be bilateral; a unilateral defect is of far more diagnostic value. Our knowledge of the mechanism of the abdominal reflexes is of the scantiest; we know that they are frequently absent when there is a lesion in the pyramidal tract; yet we have no idea why they should disappear at the very beginning of a disseminated sclerosis, and only very late in the course of an amyotrophic lateral sclerosis. That they do so behave may nevertheless be a point of considerable importance in the differential diagnosis of the two diseases.

The cremasteric reflex (L. 1, 2) may be of use where it is important to determine exactly the site of a lesion in the lower portion of the cord. The usual method of stimulation is to stroke the inner surface of the upper part of the thigh from above downwards; firm pressure in this region may elicit the reflex where gentle stroking has failed. Failure to elicit the reflex, especially if bilateral, is not necessarily indicative of disease, especially in older individuals.

The knee jerk (L. 3, 4) is the classical example of the tendon reflex. I prefer to elicit it with the patient sitting in a chair. I then place my left arm along the edge of the chair under the patient's leg, so that the foot is raised a little from the ground; the leg must be allowed to dangle loosely. A firm tap on the patellar tendon between the patella and and the tibial tubercle should produce a contraction of the quadriceps, more especially of the *vastus internus*, and after a momentary pause a secondary contraction of the hamstrings. It is the absence of this synergic contraction of the hamstrings which is primarily responsible for the "cerebellar" or "pendulum" type of knee jerk, where the leg goes on swinging backwards and forwards instead of rapidly resuming its initial position.

If there is any reason to suspect the existence of patellar clonus, first see that the quadriceps is relaxed, then grasp the patella firmly between finger and thumb and jerk it forcibly downwards.

There are a number of methods of obtaining the ankle jerk (S. 1, 2), each with its advocates. I prefer to have the patient sitting; I grasp the front part of the foot, and lift it slightly; it is essential that the patient should allow me to take the whole weight of the limb. A tap on the *tendo Achillis* then produces the reflex. My grasp of the foot serves two purposes: it assures me that the leg is relaxed, and it tells me of any weak response which my eye may not observe.

To obtain ankle clonus, with one hand I support the leg at the knee, as I did when investigating the knee jerk; with the other hand I grasp the fore part of the foot and jerk it forcibly upwards.

It is sometimes of importance to distinguish between a functional and an organic clonus, although the diagnosis of functional disease should never be dependent on this alone. Forcibly depressing the big toe will at once inhibit either patellar or ankle clonus of organic origin; but the hysteric, ignorant of this interesting fact, will go on producing his clonus no matter how hard one may depress his toe.

Probably no single method of physical examination is more often performed incorrectly than the investigation of the plantar reflex (L. 5; S. 1, 2). The instrument used may be so sharp as to cause pain; the foot may be cold, instead of being well warmed; the tendons of the foot may be tense, instead of being completely relaxed; the stimulus may be so gentle as not to produce any response at all, or so firm as to cause pain; most frequent cause of all, the stimulus may be applied in quite the wrong place.

Standing proximal to the ankle, and facing distally, take the foot firmly in the left hand, and assure yourself that there is no tension in the muscles or tendons of the sole. Then place whatever instrument you are using (I use the handle of my percussion hammer) firmly in contact with the sole of the foot at a point about 2·5 centimetres (one inch) proximal to the fifth metacarpo-phalangeal joint and directly below the fifth metacarpal bone. Allow the instrument to remain in contact with this point for a second or so before moving it; this will in many cases obviate the production of "that ticklish feeling." Then drag the instrument with a firm even pressure distally along the line of the fifth digit until it reaches the depression where the small toe begins. While you do this, watch any movements which the toes make, and pay particular attention to the first movement

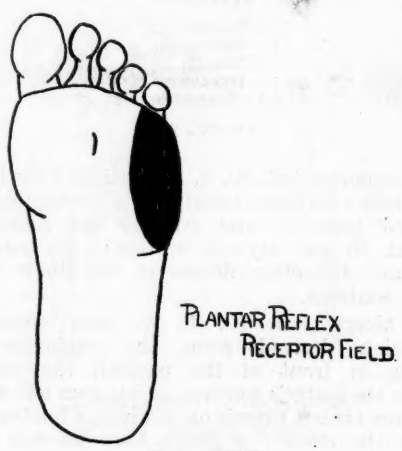


Figure XVIII.

which they make, rather than to any secondary defensive withdrawal.

The response is said to be flexor when the big toe is deflected downwards; extensor when it is deflected

upwards (in which case there is always a simultaneous contraction of the hamstrings, which should be felt for); and equivocal, when the examiner is not quite sure what is happening. I suggest to you that by the adoption of this technique the number of equivocal responses you will obtain will be greatly decreased.

The receptor field for the reflex is comparatively small (Figure XVIII); by receptor field I mean that area, the stimulation of which will first demonstrate any alteration in the character of the reflex. It is very instructive and a striking commentary on the valuelessness of attempting to elicit the reflex from any other point on the sole, to see an early case of disseminated sclerosis, in which stimulation of the receptor field will produce a definite extensor response, while stimulation anywhere else on the sole will evoke an equally definite flexor. Later in pyramidal disease, it is true, an extensor plantar may be evoked from stimulation anywhere on the sole, or even as high up the leg as the knee; but then the disease is well advanced and a golden opportunity of early diagnosis and early treatment may have been missed.

Oppenheim's reflex is not, contrary to popular belief, more sensitive than the plantar reflex correctly elicited; it may, however, provide useful confirmatory evidence. To produce it, one strokes firmly downwards with the thumb along the postero-internal surface of the tibia. The significance of the movement of the toes thus obtained is identical with that of the true plantar reflex.

Where there is any question of the precise localization of a lesion in the sacral region, the presence of a bulbo-cavernosus reflex will provide evidence of the integrity of the third and fourth sacral segments. A contraction of the *bulbus urethræ* will be felt if the *glans penis* is stimulated by pricking or pinching.

Where any reflex is difficult to elicit, reinforcement may be of value. The method I prefer is to ask the patient to place one hand on my upper arm, whichever hand is more convenient. Then, when I say "Grip," he grips and I attempt the reflex again. The general increase in tonus produced by the voluntary effort is sometimes useful in bringing out a previously latent reflex. I prefer this method of reinforcement to the better known technique, where one gets the patient to clasp his hands together and then pull; in the former one is much more certain that he really is cooperating.

That concludes the list of reflexes. Although for clearness sake I have described separately the examination of the motor system, of the sensory system and of the reflexes, it is obvious that you will save a good deal of time if, when you have the patient's arms exposed, you conduct all the necessary investigations in that region, a procedure which you will then repeat with each successive area of the body as you uncover it.

Before we finally pass from the reflexes, it may be of interest to look at one or two diagrams showing the variation from the normal produced by a few common lesions. In a peripheral neuritis there is, as you see, an all-round diminution or absence of reflexes, only the abdominals remaining unimpaired (see Figure XIX). In the ordinary hemiplegia following cerebral thrombosis, reflexes on the sound side are uniformly unaffected; on the paralysed side there is an extensor plantar response, an absence of abdominal reflexes and of the Leri and Mayer

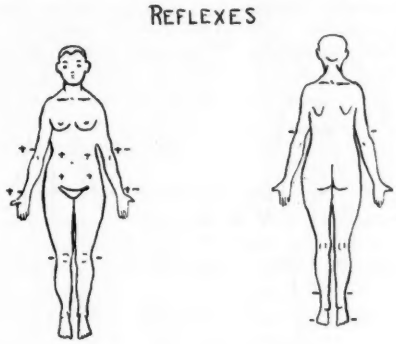


FIGURE XIX.

reflexes, a general increase of tendon jerks, and one can elicit both knee and ankle clonus (see Figure XX).

The case of extramedullary spinal tumour, the sensory charts of which I showed you just now, gave the picture shown in Figure XXI. The right side remains unaffected, and so does the left arm; but the lesion is above the segments concerned with the abdominal reflexes, which, together with the left leg, show the changes typical of a pyramidal lesion, which you saw in the preceding diagram.

The Patient's Station and Gait.

The attitude which the patient assumes, whether he be in the erect or the recumbent position, will often give you an inkling of what disease it is you are dealing with long before you have made a definite diagnosis. Since one's memory for such matters as attitudes and gaits is visual rather than auditory, I shall show you a few pictures of certain characteristic attitudes.

1. The position of the arm in a birth palsy affecting the fifth cervical root is very typical; it has, as you will remember, been compared to that of the waiter with the itching palm.

2. Where the birth palsy has been more extensive, the resultant infantile hemiplegic often adopts this attitude; flexion of arm and leg, often with athetotic movements of the arm; the foot is in the typical equino-varus position.

3. Paraplegia in flexion, everything flexed, needs no description.

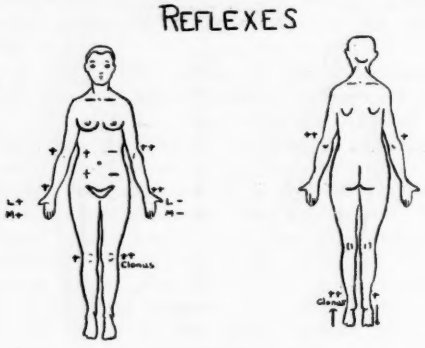
4. Hyperextension of the knees is very suggestive of tabes.

5. *Amyotonia congenita* diagnoses itself; the extreme weakness and flaccidity of the lower limbs are found in no other condition.

6. The Parkinsonian syndrome is unhappily becoming more and more familiar to us each year.

7. The extreme head retraction of a meningitis is well known to you all.

If a patient can assume the erect posture it is important to investigate the state of his equilibrium. Romberg's test is commonly used for this purpose.



LEFT HEMIPLEGIA
(UPPER MOTOR NEURONE LESION)

FIGURE XX.

A normal individual should be able to stand with his feet parallel, heels and big toes touching, and to maintain that posture, even with his eyes shut, in spite of the external interference of a few gentle pushes. In other words he should be able to maintain his equilibrium entirely without visual aid. If he cannot do this he is said to show a positive Romberg's sign. This positive Romberg's sign may be of varying degrees of severity, the mildest being when posture can be satisfactorily maintained until the body is pushed, the most severe when the patient is unable to maintain his posture even with his eyes open and his feet wide apart. It must be clearly understood that a positive Romberg's sign is of no value when the ataxia is due to muscular weakness, that is to say, in the presence of any form of paralysis affecting the lower limbs.

A positive Romberg's sign indicates some interference with the proprioceptive mechanism, and no more than that; it does not localize the lesion to any particular part of that mechanism; and I would ask you particularly to remember that it may occur in other conditions than tabes.

To get all possible information from the inspection of a patient's gait, it is essential for the legs to be exposed at least as high as the middle of the thigh; and then, when the patient walks, look at him from in front, from behind and from either side. As with postures, so with gaits; seeing them once is better than reading or hearing a description of them twenty times. Will you excuse my amateurish efforts if I endeavour to show you the outstanding features of one or two typical gaits.

1. The hemiplegic cannot actively advance his paralysed and rigid leg; he therefore moves it forward by tilting his body to the sound side, and swinging his whole body round the arc of a circle, the centre of which is the sound leg.

2. The tabetic sways a little from side to side and walks with his feet wide apart; he lifts them high, swings them forward and then lets them drop, never being quite sure when they will reach ground. The tabetic takes rather the same sort of step as the man going up an ill-lighted stairway, who carefully negotiates a top step that is not there.

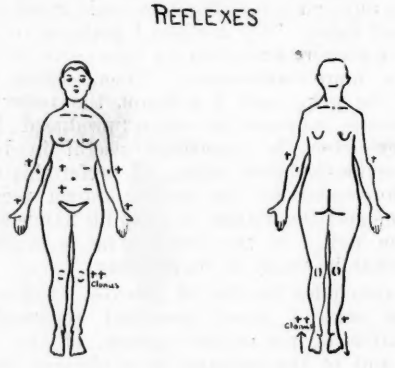
3. The patient with a mild cerebellar lesion may show no abnormality obvious at first sight; if, however, you watch his course over ten yards or so, you may notice that he is tending to deviate to one side or the other. A patient with cerebellar tumour under my observation told me that the first thing which drew his attention to the fact that something was wrong, was that his wife complained that he seemed to be trying to push her off the footpath as they walked along together. If the patient is aware of this tendency, he may consciously correct it by a strong voluntary effort, half turning his body in so doing; in other words, he walks north but faces north-east. With more advanced cerebellar disease the patient may be extremely unsteady, reeling and often falling towards the affected side.

4. The man with Parkinsonism walks like this—you all know the picture. He takes short quick steps, barely lifting his feet from the ground, but shuffling stiffly along. Practically no synergic movement takes place at any joint above the hip.

5. The hysteric can produce almost anything you can possibly imagine in the way of gaits. If ever you see a patient walking with a weird and wonderful gait, the like of which never was on sea or land, then you are probably looking at a hysteric who is secretly rather proud of his achievement.

Additional Methods of Examination.

That concludes the ordinary physical examination. My time draws short, and I can only mention briefly



EXTRAMEDULLARY SPINAL TUMOUR
(6TH LEFT THORACIC SEGMENT)

FIGURE XXI.

certain additional methods of diagnostic value in certain cases; the choice among these methods will depend on the findings of the routine examination, and also to a certain extent on the skill and experience of the investigator. I have divided these additional

diagnostic methods into two groups, primary and secondary. Each member of the secondary group requires the performance of a primary method as a necessary preliminary.

Primary Methods.

Among primary methods I include all methods of obtaining access to the circulating cerebro-spinal fluid; these are, in cranio-caudal order, ventricular puncture, cisternal puncture and lumbar puncture. Ventricular puncture, though decidedly not a procedure for the novice, is not nearly such a fearsome business as it sounds, and it is possible to obtain access to the lateral ventricle without harming the patient in any way. The site of election for the puncture is 18 millimetres (three-quarter inch) above the superior curved line of the occipital bone and 2·5 centimetres (one inch) from the mid-line. Under local anæsthesia the skull is exposed through a small incision, a tiny drill hole is made and the needle is inserted horizontally, with its point directed forwards and slightly outwards. For diagnostic purposes ventricular puncture is used as a necessary preliminary to ventriculography; therapeutically it is performed by some workers in cases of meningococcal meningitis.

On cisternal puncture, its technique and its uses, there has been a great deal written in recent years; suffice it to say that the method has established itself as of both diagnostic and therapeutic value, and that in experienced hands it is certainly no more hazardous a procedure than the more customary lumbar puncture. You will remember that one introduces the needle in the mid-line of the neck, just below the lower border of the occipital bone, and in the line of the external auditory meatus and the outer angle of the eye. The distance to the medulla in an adult is roughly seven centimetres; I therefore use a needle only six centimetres long in order to be on the safe side.

Concerning lumbar puncture I have only two small pieces of advice. The first, never carry out lumbar puncture on a patient with disseminated sclerosis, if it is in any way possible to make a diagnosis without so doing; if you do puncture such a patient, your reward may be to have to deal with a headache lasting for weeks on which morphine in large doses has about as much effect as distilled water. The second, always keep patients on whom lumbar puncture has been performed in a recumbent position for at least twenty-four hours after the puncture has been performed; you will thus escape practically all the unpleasant sequelæ that may follow on this operation.

The other diagnostic methods which I have classified as primary are radiography and the taking out of electrical reactions. Of the assistance which an X ray picture of the skull or of the spine may give to the neurologist there is little need to speak; calcified suprasellar cysts, tumours involving the skull, tumours involving the vertebræ, are only a few of the many conditions in which the cooperation of the radiologist is invaluable. In such procedures as ventriculography, encephalography, and "Lipiodol" injection, the taking of an X ray picture is, of course, an integral part of the method and requires no special mention here.

On the subject of electrical reactions I shall be very brief; I have never used them; I have never seen a diagnosis made by their aid. I cannot avoid a feeling of suspicion that those authors who give these reactions such a prominent place in their works on neurology do so partly because they are unwilling to break with an old tradition handed down from text book to text book. I suspect further that in their own practices these gentlemen make perhaps very little more use of electrical reactions than we humbler mortals do.

Secondary Methods.

The secondary methods of investigation depend upon the establishment of communication between the circulating cerebro-spinal fluid and the outside world.

The taking of an accurate pressure reading should be a routine part of every lumbar puncture. I cannot emphasize too strongly how absolutely unreliable and misleading is any estimate of intrathecal pressure based on the manner in which fluid flows from the needle. If the pressure is worth commenting on at all, it is worth recording accurately; and this is such a simple procedure, involving no technical skill whatever beyond that required to introduce the needle into the theca, that I cannot understand why it is not more widely known and practised. I use, and find very convenient, a Greenfield needle, fitted with a three-way tap; to this is connected, by means of a short length of rubber tubing, a glass tube thirty centimetres (a foot) long, graduated in millimetres. The needle is introduced in the ordinary way; as soon as you think it is in the theca, withdraw the stilette a little; as soon as a drop of cerebro-spinal fluid appears at the base of the needle, withdraw the stilette completely and turn the tap upwards. The fluid flows into the glass tube, and one reads the pressure direct in millimetres of cerebro-spinal fluid.

It is advisable at this stage to perform the Queckenstedt test. An assistant manually compresses the internal jugular vein on one side, and this compression in normal cases will produce a rapid rise in the cerebro-spinal fluid pressure, followed by an equally rapid fall as soon as the compressing hand is removed. Failure to elicit this rapid rise and rapid fall is highly suggestive of a block somewhere in the spinal subarachnoid space, such as is produced by a spinal tumour.

The fluid which is in the manometer may then be allowed to escape into a sterile test tube by turning the tap towards the base of the needle. When five cubic centimetres of fluid have been thus collected, it is advisable to turn the tap back to the vertical position again, and to see what effect the removal of this amount of fluid has had on the intrathecal pressure. A very considerable drop from the initial pressure, especially if coupled with absence of the Queckenstedt response, is strong evidence of loculation in the spinal subarachnoid space. The normal intrathecal pressure varies from 100 to 170 millimetres of cerebro-spinal fluid.

Reports of pressure readings may be made as shown in the accompanying table. I would draw your particular attention to the figures in this case of cerebral tumour. It is a golden rule, when cerebral tumour is suspected, never to reduce the pressure below two-thirds of the initial reading; hence you will withdraw small quantities of fluid at a time, and take pressure readings after each such withdrawal.

MANOMETRY IN LUMBAR PUNCTURE.

	Normal.	Spinal Block.	Cerebral Tumour.
Initial Pressure	150	160	270
Right Jugular Compression:			
Rise	250 in 5″	180 in 15″	300+ in 3″
Fall	150 in 4″	170 in 15″	270 in 4″
Fluid Removed	5 c.c.	5 c.c.	3 c.c.
Final Pressure	110	60	200

The fluid thus removed is subjected to certain routine examinations; it is far too late to do more than simply to enumerate these.

The examinations which I perform on every fluid are: (i) Note the appearance, (ii) make a cell count, (iii) estimate the total protein, (iv) estimate the globulin, (v) estimate the sugar, (vi) estimate the chlorides.

Where they are called for, the following methods may give additional information of value: (i) Carry out a Wassermann test, (ii) plot a Lange gold curve, or better, because easier to perform, the bicoloured guaiac curve of De Thurzo, (iii) estimate the urea.

Ventriculography consists in the performance of a ventricular puncture, of which we have already spoken, the removal of as large a quantity of cerebro-spinal fluid as is possible through the drill hole, and the replacement of this fluid by a somewhat smaller amount of air. A radiogram is then taken to show the air lying in the ventricles, the object being to determine if there is any deformation of these chambers by a tumour lying in or near their walls. The whole procedure may give valuable information, but it is not without its own operative mortality; it is, in my opinion, a job for the neurological surgeon rather than for the neurological physician, because a sudden rise of intracranial pressure may occur necessitating immediate surgical decompression, and this is a contingency which should never find the operator unprepared.

Encephalography is a somewhat similar procedure, but here the air is introduced through a lumbar puncture needle into the spinal subarachnoid space. The injected air passes cranially from the site of injection towards the medulla; some of it may pass through the foramina of Magendie and Luschka into the fourth ventricle, and so ultimately into the lateral ventricles. For filling these ventricles, however, it is not nearly such a reliable method as ventriculography. The remainder of the injected air will pass out in the cerebral subarachnoid space over the surface of the hemispheres, and will outline any block present in the space; it was for this purpose that the method was introduced by Dandy. The whole procedure, while fairly simple to perform, is apt to give rise to a most distressing headache, comparable to that produced by the lumbar puncture of a case of disseminated sclerosis; I do not recommend it unless there is no other way of arriving at a diagnosis.

Of the injection of "Lipiodol" into the *cisterna magna* in suspected cases of spinal tumour I have already spoken earlier; it remains only to show you pictures of cases where this method has been used successfully.[1]

Conclusion.

That concludes my description of a scheme for the examination of the nervous system. It has not been possible to discuss the interpretation of the results one may obtain in the carrying out of this scheme. After all, such interpretation forms the greater portion of the huge structure of neurology, of which structure we are tonight only dealing with the foundations; yet without these foundations the whole structure must fall to the ground.

Although I have already talked far too long, I should like to say one more word, before I close, in defence of neurological therapy. There are probably still a number of gentlemen in this hall who very much doubt whether it is worth while, from the patient's point of view, going to all this trouble to make an accurate neurological diagnosis. If it should ever come about that we debate this question, I warn these gentlemen that I shall bring with me a veritable host of witnesses. There will be my friend with the cerebral tumour of whom I spoke to you; there will be the patient with spinal tumour, who after being confined to his bed for a year, now walks, runs and swims; there will be the patient with subacute combined degeneration of the cord, vastly improved by liver therapy; there will be the cured meningitic, saved by timely cisternal puncture from death and that chronic hydrocephalus which may be worse than death; there will be the epileptic whose fits are only a memory of the past; there will be the patient with general paralysis of the insane, now, after a course of malaria, an active and useful member of society again. My own evidence, gentlemen, you may not believe; but the evidence of my witnesses you will find it hard indeed to refute.

I thank you again for your courtesy in giving me the privilege of speaking to you this evening, and for your kindly patience in bearing with me so long.

Reports of Cases.

PYOGENIC VERRUCOSE DERMATITIS.

By George R. Hamilton, M.B., Ch.M. (Sydney),
Honorary Assistant Dermatologist, Sydney Hospital; Honorary Dermatologist, Royal North Shore Hospital; Honorary Dermatologist, Royal South Sydney Hospital,

and

E. B. Jones, M.B., Ch.M. (Sydney),
Clinical Assistant, Department of Pathology, Sydney Hospital.

Clinical History (G. R. Hamilton).

Although I have since seen several cases of pyogenic verrucose dermatitis, it was not until the almost dramatic disappearance of the condition in my first case that I

[1] At this stage Dr. Hunt showed some skiagrams demonstrating "Lipiodol" in the spinal canal.

fully realized the importance of Dr. Norman Paul's article on this disease in THE MEDICAL JOURNAL OF AUSTRALIA of February 25, 1928, page 240.

The patient, J.S., a male, aged fifty-four, a painter, noticed a small pimple on the dorsum of the right hand between the fourth and fifth metacarpal bones. This became broken and the site later became inflamed. The inflammation spread concentrically and later the condition became verrucose in appearance.

On examination the lesion was seen to extend from the wrist practically all over the dorsum of the right hand for about two centimetres on to all the fingers. The lesion was somewhat raised, more so at the edges, and scattered over the area were islands of necrotic skin, mosaic-like in appearance and discharging on pressure some scanty pus.

Thinking the condition was one of tuberculous verrucose dermatitis, I treated it accordingly for quite a long time with no result. Examinations were made for yeasts, bastomycosis *et cetera* with a negative finding. The patient gave a positive Wassermann reaction and was undergoing treatment for syphilis during the time he had his dermatitis.

A section was submitted for histological examination and later pus was cultured, when a non-hæmolytic streptococcus was grown from which a vaccine was prepared. It was then that I decided the condition was similar to the one Dr. Paul described, and I handed the patient over to Dr. Jones for antistreptococcal treatment.

HISTOLOGICAL REPORT AND PROGRESS (E. B. JONES).

A portion of tissue was removed from the dorsum of the hand for microscopical examination.

The portion of tissue examined showed most intense inflammatory changes. Neutrophile leucocytes were present in great numbers, and in many places there were areas of necrotic and abscess cavity formation to be found. The skin surface itself in many places was ulcerated.

On microscopical examination no definite evidence of tuberculosis or syphilis was to be seen. There were many abscesses in the thick epidermal network. Cultures were made from the discharge and a streptococcus was isolated.

Three doses of antistreptococcal serum (Commonwealth Laboratory) were given intramuscularly, the first day ten cubic centimetres, followed five days later by fifteen cubic centimetres, and in another five days twenty cubic centimetres. The patient then developed serum sickness and was not seen for a week. When he returned, the lesion was greatly improved and continued to improve for the next two weeks, when autogenous streptococcal vaccine therapy was commenced. The vaccine had been prepared in series containing five, fifty and five hundred million organisms per cubic centimetre. The first dose was 0·06 mil (one minim) of the first series and this was gradually increased until one cubic centimetre of the last series of five hundred million organisms was injected.

On the completion of the vaccine treatment the lesion had completely healed.

Reviews.

DYSPEPSIA VIEWED BY A SURGEON.

THE second edition of "The Surgical Dyspepsias," by Walton, is chiefly an index of the further experience of the author during the period of seven years since the publication of the original work.[1] The title of the book partakes of the nature of a slogan, to emphasize the fact that surgery does play a part in the treatment of dyspepsia.

The reader will find that not only is the home of dyspepsia directly concerned, but also that most of the abdominal organs are exhibited in some sort of ætiological relationship. Diseases of the gall bladder, pancreas and appendix are shown to play their various parts, whilst ptosis of the different units of the alimentary canal is dealt with in a special chapter. With his two- or three-fold number of cases since the publication of the first edition there is little variation in the author's original ideas. He finds that on further experience gastric ulcer is not associated with such a high percentage of instances of hyperchlorhydria, and that most of his previous percentages of symptoms, signs, tests *et cetera* are maintained. We think at times, when reading the book, that it might have been more entertaining if the statistics had been relegated to a special appendix. A follow-up system has been adopted with the result that many methods of treatment have been more correctly gauged. Naturally, in a book of this kind prominence is given to gastric and duodenal ulceration, the causation of which is discussed, with a verdict in favour of bacterial invasion.

The vexed question of medical *versus* surgical treatment is settled in the manner expected of a specialist in surgery after some consideration of the views of McLean and Hurst. Restriction of operation to suitable cases makes for favourable statistics, whilst in medically treated cases a life-long special dietary is emphasized. Acute ulcer is practically a medical disease.

Technical details of the various operations are shown in their relationship to after effects. Apparently there is still a lack of finality with regard to the best methods to be adopted in the treatment of ulcer. Excision or cautery of ulcer, pyloric occlusion *et cetera* as auxiliaries to gastro-enterostomy are fully discussed. Other diseases of the stomach, from inflammatory to malignant, receive well balanced consideration. The interesting subject of duodenal ileus receives more ample attention than in the first edition, the diagnosis being almost entirely dependent on X ray examination.

The latter half of the book is devoted to diseases of the liver, gall-bladder and bile ducts, to diseases of the pancreas and of the appendix. Appendix dyspepsia has fitting space devoted to it and no doubt exists in the mind of the author with regard to the causative factor in these cases.

Finally, and with many warnings and some reluctance, the various ptoses of the viscera are dragged by the author into the surgical arena.

Whilst lacking the charm of Lord Moynihan's writings, the book is one of the most comprehensive that has been founded on English experience and contains the matured opinions of an eminent surgeon of wide practice.

The second edition is no larger than the first. The illustrations on the whole are good. We can recommend the book as a sound practical work on the subjects that have received consideration.

SERMONS AS ESSAYS.

A BOOK both stimulating and entertaining is Professor Murdoch's collection of essays, entitled "Speaking Personally."[2] We feel ourselves fortunate in being able, through these pages, to make the acquaintance of one so charming, witty and wise, whose outlook is so fresh and

[1] "A Text-book of the Surgical Dyspepsias," by A. J. Walton, M.S., M.B., B.Sc., F.R.C.S.; Second Edition; 1930. London: Edward Arnold and Company. Royal 8vo., pp. 728, with illustrations. Price: 42s. net.

[2] "Speaking Personally," by Walter Murdoch; Third Edition; 1930. Sydney: Angus and Robertson. Crown 8vo., pp. 208. Price: 6s. net.

whose interests are so various. The author is a self-declared "preacher," a fact that he humorously deplores in the preface, warning us against his vicious propensity to point a moral and giving us permission to "skip the pontifical passages." We must confess, however, that if all preachers had such charm and persuasiveness, they would be sought rather than shunned and our natural dislike of being morally bettered would quickly be conquered. Beneath a light whimsicality of manner we find philosophical reflections of no mean calibre and which bespeak the author's seriousness of purpose. He wages war fiercely against what he chooses to call "the suburban spirit," that greatest of all enemies, which kills our minds and souls and makes us "degenerate into poor spiritless conforming creatures"; let us rather be vagabonds and free ourselves from the trammels of conformity. It is this idea which pervades the essays. Though there is much to be said for convention as a bulwark against social chaos, we are apt to mistake comfort for civilization and respectability for morality; few of the world's greatest thinkers were considered respectable in their day.

The title, we are told, is by far the most important and the most difficult matter in the art of essay writing, nor must an attractive title be affixed to a dull essay; this, in the author's opinion, would be a "base act of treachery." However it may be, these essays are fraught with many surprises. For instance, an essay "On Trousers" turns out to be a plea for nationality. "Thoughts on a Gargoyle" is not a discussion on this product of mediaeval architecture, but a philosophical consideration of our conception of heaven and hell, of perfection and imperfection, winding up with an appeal to Australian political leaders for idealism. "On Sitting Still" begins by telling us of a new religion known as "Seizaho" or "Method of Quiet Sitting," the practice of which is claimed to be infallible in "strengthening the stomach, intestines and sense of justice." The essay thus far is delightful in its absurdity; but the author has yet a far more important matter to lay before us and proceeds to preach the "gospel of leisure," urging us to "loaf and invite our souls" by the serious business of thinking. "On Chewing Gum" is a sparkling satire on modern America, undeniably pre-eminent "in breakfast foods, tar roofing, culture, office furniture, righteousness and chewing gum." "Are You Stock Size" is an appeal for intellectual honesty: "Be honest with yourself and recognize that your mind is dressed from top to toe in reach-me-downs." The word "bloke," absurd and lacking in dignity as it is, expresses for the author all that democracy stands for, the true brotherhood of man. He goes on to remark profoundly that equality must essentially mean "equality in capacity to suffer." And so on; the topics dealt with are endless in their variety and the author's judgements are always sound.

Perhaps the secret of Professor Murdoch's charm is that he speaks, not as a superior being from heights immeasurable, but as one of us—in fact, as one "bloke" to another. This informality and intimacy of tone, allied to the vividness and sparkle of his style, his shrewd wisdom and his genial satire based on so keen a sense of the comic, make him a delightful writer and one who is never dull. In spite of the author's apologies in the event of having "done us good," we feel refreshed.

Analytical Department.

"VITAB."

"VitaB" is an Australian product consisting of pure wheat germ obtained from first grade wheat, the summer crop of the Riverina district, New South Wales, being the source. By a patented process the germ is mechanically freed from all the other constituents of the grain. The method of preparation insures a final product of uniform purity and consistency. When the germ has been obtained in this pure form it is transferred in clean bags from the country mills to Sydney.

In the Sydney laboratory the product is treated in an electrically heated apparatus, so constructed that a uniform temperature of its contents is assured. A temperature not exceeding 70° C. for twelve minutes is used. The product is then packed straight from the apparatus into paper wrappers in one pound tins. The whole process, both in the country mill and city laboratory, is carried out in a thoroughly hygienic manner and the material is not touched by hand at any stage.

The final product is attractive in appearance and of pleasant taste. It is to be eaten without any further treatment, and can be added to a variety of foods, such as porridge, cereals, stewed fruits *et cetera*, or can be taken with warm milk and sugar.

The process of manufacture is such that a guarantee can be given that the original vitamin content of the germ is present in the product as marketed.

Wheat germ contains the following vitamins:

Vitamin *A*. This is present in large amount and probably in direct relation to the yellow colouring matter.

Vitamin *B*1. The antineuritic and growth-promoting vitamin is present in large quantities. It is probably the richest natural source.

Vitamin *B*2 (Vitamin *G* or *P.P.*). The pellagra-preventing and growth-promoting vitamin is also present in large amounts.

Vitamin *E*. The anti-sterility vitamin. This is the richest known source of this vitamin.

Analysis.

As marketed, the substance consists of deep yellow flakes from one to two millimetres in diameter, each flake being the pressed germ of one wheat grain.

Two samples of different batches have been analysed with the following results:

Constituent.	Sample 1.	Sample 2.	Average.
Protein	30·3%	30·5%	30·40%
Fat	11·4%	9·7%	10·55%
Moisture	9·0%	8·0%	8·50%
Ash	4·6%	4·5%	4·55%
Carbohydrate (by difference) ..	44·7%	47·3%	46·00%

Vitamin *B*1 is relatively heat labile, but according to the experimental work of McCollum and Davis, and of Chick and Hume, it is able to resist a temperature of 100° C. for two hours without being destroyed. As "Vita B" is in sterilization only subjected to a temperature of 70° C. for twelve minutes, there can be no destruction of vitamin *B*1. The other vitamins are heat-resistant.

Biological experiments on young rats have been in operation for over a month and exceedingly satisfying growth results have been obtained. A full and critical account of these experiments will be given in about two months' time. Up to date it has been proved that 0·5 gramme of "VitaB" daily, added to an otherwise vitamin *B* free diet, determines a steady increase of growth at a somewhat lower rate than that of a control animal on a mixed diet, but 2·5 grammes of "VitaB" daily determines a growth rate much above that of the control animal.

The manufacturers have certified to us that the experimental "VitaB" product that was delivered as samples to the medical profession, is no longer being produced by them and that such tins of this experimental product that were offered for sale by chemists have all been withdrawn. The only product now being manufactured by them and offered for sale by chemists conforms to the description in this article.

"VitaB" administration would be of value in any condition in which a high vitamin diet is indicated. It should be of value wherever white flour enters largely into the diet and there is a lack of fresh fruit and vegetables. This would hold especially in the case of under-nourished children. As there is practically no storage of vitamin *B* in the body, its administration would seem to be indicated whenever the diet, through illness or other cause, has been deficient in adequate vitamin *B*. It should prove of value in pregnancy (Mellanby). It may also be found of value in the treatment of certain forms of constipation, partly owing to its cellulose content, partly owing to the tonic effect of vitamin *B* on the alimentary tract.

The Medical Journal of Australia

SATURDAY, APRIL 18, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ASTHMA.

Until a comparatively recent date, when the conception of allergy was introduced, very little that was new had been written about asthma for many years. With the introduction of skin tests and the discovery that the patient was sensitive to specific proteins, hopes rose high not only of relief, but of cure for many sufferers. These hopes were not realized, for the percentage of patients whose condition was ameliorated was not very high. At the same time, allergic investigations have been continued and biochemists have supplied data which may prove to be of fundamental importance.

In endeavouring to form an estimate of the present state of knowledge of the nature and causation of asthma, the first point to be considered is that of diathesis. It would seem to be impossible to avoid the postulation of a congenital constitutional abnormality in asthmatics. A. F. Hurst is one of those who believe in the existence of an asthma diathesis. Speaking at the Annual Meeting of the British Medical Association in 1929, he said that this diathesis is caused by a slight deviation from the average blood chemistry which results in the vagal constituent of the bronchial nervous system becoming the predominant partner. He thought that in asthmatics certain chemical, reflex and physical stimuli which have no effect on normal individuals give rise to the asthmatic attack. J. André believes that asthma is caused by a diathesis analogous with the colloidal diathesis of Widal; he defines this as "the state created by toxæmia of all kinds, autogenous or exogenous, alimentary or infective, protein or mineral." It is difficult to understand how a congenital state can be analogous to one created by toxæmia. However this may be, most investigators will agree that some type of diathesis may be presumed to exist. It will, of course, be stated at once that allergy is the manifestation of a congenital peculiarity and that allergy furnishes the explanation of many forms of asthma. This may be granted, but it must be remembered that there is a difference between asthma which is definitely allergic and so-called non-allergic asthma. In allergic asthma the skin gives a reaction to one or more proteins, eosinophilia is present and desensitization may often be carried out by injection of the offending protein. In so-called non-allergic asthma, sensitization cannot be determined, eosinophilia is absent and the cause may be of a reflex nature. The term "so-called" non-allergic asthma is used because it is quite obvious that a sensitized state of all protoplasm might exist which has not been discovered. It is also conceivable that other factors may exist in addition to an asthmatic diathesis and a stimulus, be that stimulus nervous impulse or of protein origin.

If an asthmatic diathesis be accepted, it is necessary to discover the factors determining the onset of asthmatic attacks. This is primarily a matter for the physician, who will have to call to his aid such persons as the rhinologist, the biochemist and the bacteriologist. The complexity of the investigations is shown by a recent report on the work of the Asthma Clinic at Guy's Hospital, published in *Guy's Hospital Reports* for October, 1930. This is not the place for a lengthy description of such work, but it may be pointed out that two hundred patients were investigated from the general medical, immunological, bacteriological and biochemical standpoints. Perhaps most of the newer work has

been along biochemical lines. Much of this has been discussed on previous occasions in these pages. Reference has also been made to the work carried out at Guy's Hospital in the isolation from the urine of a nitrogenous substance, a proteose, to which the patient gives a positive skin reaction. No more is needed to indicate the complex nature of the asthma problem and the different points of view from which it must be attacked.

The expedient adopted in certain parts of the world for the elucidation of the asthma problem has been the establishment of special clinics at which patients may be treated and subjected to continuous observation and study; the clinic at Guy's Hospital is an excellent example. The difficulty and the magnitude of the problem have justified this procedure. When similar steps are taken in Australia (and in some places such steps have been taken) several facts must be borne in mind. The first is that, though the treatment of the individual patient is important, the elucidation of the factors producing his malady is much more important. It follows from this that each patient must be subjected to more than what might be called a symptomatic investigation. For example, if it is found that an asthmatic individual is sensitive to a certain pollen, investigation should not end with this discovery, nor should the medical attendant be satisfied with giving a series of protective injections. Every avenue of investigation must be explored and the findings must be recorded for correlation with future findings. It is evident that such a clinic will be more than an "allergy clinic." The next point of importance is that investigators belonging to all branches of medicine bearing on the subject should be attached to the clinic and that each patient's condition should be subjected to the scrutiny of the whole staff. The final point is that, if possible, experimental investigations on animals should be carried out. When work is organized on these lines we may possibly unravel the tangle of all the factors likely to produce asthmatic attacks and discover the true nature of the asthmatic diathesis. Then and not till then can the ideal be reached—the preventive and not the curative treatment of asthma in all its forms.

Current Comment.

PAROXYSMAL TACHYCARDIA.

Since the publication of Richard Payne Cotton's article in 1867, paroxysmal tachycardia has been an interesting clinical entity whose causation was attributed to failure of the inhibitory fibres of the vagus or over-stimulation of the sympathetic nerves, with a neurotic or hysterical predisposing factor. It is only in comparatively recent years that it has been scientifically investigated by such instruments of precision as the electrocardiograph and Mackenzie's ink polygraph. W. E. Hume[1] gives an excellent account of the manifestations of this disorder. It may be classified as auricular, auriculoventricular or nodal and ventricular. Auricular tachycardia may be again divided into regular auricular tachycardia, auricular flutter and paroxysmal attacks of auricular fibrillation. If the tachycardia be generated in the vicinity of the sino-auricular node, the impulse will pass to the ventricles by the normal paths and the time of passage will be approximately normal. If the auricular rate be very great (for instance, 300), the ventricles may respond only to some of the auricular impulses, constituting auricular flutter. In regular auricular tachycardia, the auricular and ventricular rates are the same and vary from 140 to 250. An attack may sometimes be checked by pressure on the vagus in the neck. Whether this is effectual by stimulation of the vagus itself or the nerve of Herring is not clear. In auricular flutter vagal compression does not arrest the attack, but may slow the ventricular rate for a time. Auriculoventricular or nodal tachycardia is the most common form. In the superior nodal type the electrocardiogram suggests that the excitable centre lies in the upper part of the junctional tissues, and the wave of excitation spreading towards the auricles and ventricles, excites the auricles first. In the inferior nodal it would appear that the excitable centre lies in the lower part of the junctional tissues and the wave of excitation excites the ventricles first. If the excitable centre be midway between auricles and ventricles, the wave of excitation passes simultaneously upward and downward, and there will be simultaneous contraction of auricle and ventricle. This is termed by Hume the nodal subdivision, which rather confuses the nomenclature. In ventricular tachycardia the paroxysm originates in either ventricle. The ventricular complexes have the characteristics of extrasystoles from right or left ventricle. In the intervals between the paroxysms premature ventricular beats may occur with complexes like those of the paroxysm. It can often be shown that the beginning and end of the paroxysm have the same relation to the slow rate as do the premature extrasystoles to the normal beats. This form of tachycardia is more often associated with the more serious forms of heart

[1] *The Lancet*, November 15, 1930.

disease, particularly coronary thrombosis. In many instances of ventricular tachycardia digitalis medication has been prolonged in considerable dosage and, possibly, ventricular tachycardia may be a toxic effect of digitalis where organic heart disease is present. Ventricular tachycardia has been rarely observed in the young and in persons without obvious cardiac disease. Some observations suggest that foci in both ventricles alternately emit rhythmical impulses, but an alternative explanation is that the right and left branches of the bundle of His are alternately refractory and that supraventricular impulses pass down one or other branch alternately.

The type of paroxysm remains constant to the individual, but there may be mixed types. In one instance the exciting centre seemed to move from the sino-auricular node to the auriculo-ventricular node. In another instance auricular paroxysmal tachycardia was interrupted by an attack of ventricular tachycardia. Generally in the supraventricular varieties the same type of paroxysm persists year after year, but during an attack partial heart block or a bundle branch block may appear and alter a previously regular rhythm. Supraventricular types are by far the most common, the nodal type being the most frequent.

Paroxysmal tachycardia is differentiated from all other kinds of palpitation by its abrupt onset and abrupt termination. Prodromal symptoms are generally absent and the patient is taken unawares.

During an attack the heart rate is usually between 150 and 260. It is not often above or below these figures. Regular and prolonged ventricular rates of about 150 may exist with a 2:1 auricular flutter, the auricular rate being about 300. During an attack the rate continues fairly constant; but in prolonged seizures there may be greater variation. A ventricular rate of 300 is rare. In tachycardia of supraventricular origin there is in the majority of instances no evidence of organic cardiovascular disease, but, in ventricular tachycardia, cardiovascular disease occurs in four-fifths of the cases. The heart may show no dilatation during an attack, or it may be increased up to 2·5 centimetres. If the attacks are prolonged up to four or five days, especially if organic lesions be present, the apex may move conspicuously outwards. Organic heart murmurs may disappear during an attack and congestive failure in the lungs may or may not be evidenced. Anginal pain is often felt or merely a sensation of oppression. A feeling of discomfort or numbness or pain may be experienced in the upper limb. The arterial pressure generally falls during an attack owing to diminished systolic output from the shortened diastole, or diminished contractile power of the ventricles, or, in nodal tachycardia, diminished ventricular output owing to simultaneous contraction of auricles and ventricles. In rare instances arterial blood pressure rises in an attack. The venous pressure may or may not rise, depending on the occurrence or not of cardiac incompetence. As regards blood gases, there is marked diminution of arterial oxygen saturation without demonstrable pulmonary congestion. There is also low oxygen saturation of venous blood. During a paroxysm the carbon dioxide of the arterial blood is low.

Some patients are quite prostrated during an attack and must rest recumbent. Most, however, sit rather than lie down. Some patients are able to get about, even to walk up steep inclines. There may be faintness or giddiness or actual vertigo, temporary blindness or syncopal attacks, even epileptiform seizures. Aphasia, hemiplegia and gangrene of the left arm have been noted. The attack may end abruptly, but irregularities may continue before complete return to normal rhythm. Most individual attacks last less than twelve hours, rarely more than ten days. A case is recorded lasting fifteen months, but in apparently protracted attacks it is possible that temporary intermissions may have occurred. The frequency of the attacks varies greatly. Only one may take place. The frequency of seizures is often in inverse ratio to the severity of the attacks. A definite pathological lesion of the cardiac musculature has not been demonstrated.

Diagnosis is aided by electrocardiograms. A regular tachycardia will conform to the electrical features of a regular auricular flutter, for instance, 2:1—a supraventricular or a ventricular tachycardia. If the ventricular rhythm be very rapid and irregular, the probability is either paroxysmal auricular fibrillation or auricular flutter. When the response of the ventricles to the auricular stimulation in auricular flutter keeps altering, the irregularities ascertained by the stethoscope may resemble auricular fibrillation. If the ventricular rhythm be very rapid and regular, diagnosis will lie between a regular auricular flutter, a supraventricular tachycardia and a ventricular tachycardia. Vagal pressure may help. If the attack be stopped, it is certainly supraventricular; but, if there be no response to the pressure, it may be either ventricular or supraventricular tachycardia. In the heart failure of coronary thrombosis, ventricular tachycardia is a serious complication, but the final prognosis depends on the amount of coronary obstruction rather than on the superadded tachycardia. Ventricular tachycardia is uncommon except as a terminal event in serious organ heart disease, which is generally fatal within a few months.

Much has been written in recent years concerning paroxysmal tachycardia. The Bradshaw Lecture delivered by Hume is a notable contribution, enlightening the perplexities of the subject. His use, however, of the term "nodal" as a subdivision of the "nodal" type of tachycardia is unfortunate. No mention is made of the *pulsus alternans* which sometimes occurs during an attack, nor of the fulminating attacks, in which marked and rapid dilatation of the heart occurs, with a greatly enlarged and pulsating liver, cyanosis and œdema of the face and legs. In these cases death appears imminent, but rapid and dramatic recovery often follows.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Pathology of Hypertrophic Charcot's Joints.

E. S. J. King (*The British Journal of Surgery*, July, 1930) discusses the pathology of hypertrophic Charcot's joint which he considers is a grotesque form of osteoarthritis, the exaggeration of the processes concerned being due to the frequent traumatism permitted by the anæsthetic conditions of the bones and joints. The connective tissue cells undergo anaplasia to a primitive type with subsequent differentiation in various directions. Microscopically these phenomena may even suggest neoplasia. He throws out the suggestion that the stimulus of this anaplasia is the product of dissolution of small pieces of necrotic bone.

Syphilitic Coronary Arteritis.

A. R. Moritz (*Archives of Pathology*, January, 1931) has discussed the subject of syphilitic coronary arteritis. He bases his remarks on eight cases in which syphilitic aortitis was accompanied by stenosis or obliteration of one or both coronary arteries. In six the stenosis of the coronary arteries was due wholly or in part to syphilitic arteritis of their proximal portions. The author points out that the syphilitic inflammation of the coronary is not limited to that part of the artery included in the wall of the aorta. Coronary endarteritis occurs independently of aortic valvulitis and in some instances is the immediate cause of death without preceding cardiac decompensation. In two of the cases reported by the author the coronary stenosis was so complete and was of such long duration as to necessitate the hypothesis of a collateral blood supply, presumably through the Thebesian vessels. The cause of failure of the collateral circulation was not demonstrated.

Melanosis Coli.

M. J. Stewart and E. M. Hickman (*The Journal of Pathology and Bacteriology*, January, 1931) discuss the peculiar form of melanotic pigmentation of the mucous membrane of the large intestine, known as *melanosis coli*. It is due to the deposition of melanin-like pigment within the large mononuclear cells in the mucous membrane of the gut. It is generally limited to the large intestine and appendix and the colour ranges from grey or buff to brown and inky black. The chief causative factor is intestinal stasis. The more extreme grades of the condition are of comparatively rare occurrence, but if the slighter forms are included, the incidence is fairly high. The author has found the slighter manifestations present in 11·2% in an unselected series of 600 autopsies. Melanosis is a frequent accompaniment of carcinoma of the colon. The author discusses the views of different observers as to the mode of formation of the pigment. The most acceptable in his opinion is that advanced by Pick. According to Pick, aromatic protein disintegration products are absorbed from the colon and converted into melanin within the connective tissue cells by the action of a tyrosinase-like ferment.

Malignant Nephrosclerosis.

Paul Klemperer and Sadao Otani (*Archives of Pathology*, January, 1931) write about the malignant nephrosclerosis of Fahr. They have observed fifty-eight cases of renal insufficiency and in sixteen the findings were those of malignant nephrosclerosis. The most pronounced change found on microscopical examination of the kidneys was severe vascular damage. The interlobular arteries were extremely narrowed by a cellular intimal proliferation and necrosis of the arterioles was present in every instance. In the glomeruli there were found: (i) Collapse of the capillaries and anæmia, (ii) degenerative changes in the capillary wall and its epithelial lining, (iii) epithelial proliferation and occasional leucocytic accumulation. Acute alteration of the tubular parenchyma was always evidenced by fatty infiltration, hyaline degeneration and necrosis. In their summary the authors point out that essential hypertension with renal insufficiency is associated either with: (i) The slowly progressing type of atherosclerosis manifesting a gradual constriction of the vascular bed and subsequent pronounced destruction of the functioning parenchyma, or (ii) a more rapidly developing vascular change in which severe renal atrophy is absent. The latter form, the malignant renal phase of hypertension, must be divided on a pathological basis with regard to the nature of the vascular lesions into an accelerated atherosclerotic and an arteritic form. The rapidly developing obliteration of the vascular bed is responsible for the sudden onset of fatal renal insufficiency. The ætiology of the vascular condition is unknown. Possibly a constitutional or acquired angiospastic factor plays a determining rôle in producing the accelerated atherosclerotic form. The arteritic form is most probably due to the effect of toxins on vessels that have already suffered a simple degenerative atherosclerosis.

Peripheral Nerves and Squamous Epithelioma.

W. Martynow (*Virchow's Archiv für Pathologische Anatomie und Physiologie und für Klinische Medizin*, October, 1930) has investigated the peripheral nerves in their relationship to squamous epithelioma. The material for his investigation was obtained from epitheliomata of the lower lip, the penis, the tongue and the upper lip, removed at operation. He states that the nerve fibres found in human squamous epitheliomata are naked axis cylinders which have an arrangement similar to that seen in normal squamous epithelium of several layers. The majority of these nerve fibres are freshly formed. In the epithelial cell nests nerve fibres are also found; they vary with the cornification of the cells. In the connective tissue-like cancer stroma pseudonodules arise by overgrowth of nerves. Cells of Langerhans also appear in the tumour and both their bodies and their processes increase in size. In the normal epithelium in the vicinity of the tumour the intraepithelial nerve fibres are increased in number. The nerve fibres more remote from the tumour are normal. In the tissue distant from the tumour no changes are found, but in that situated somewhat nearer both destructive changes and changes of the nature of new growth are seen in the nerve fibres.

Schilder's Disease.

J. R. Perdrau (*The Journal of Pathology and Bacteriology*, October, 1930) describes the pathological appearances of the brain of a *rhesus* monkey which had died of Schilder's disease. No lesions were detected in the brain and spinal cord by macroscopical examination. Histological examination, however, revealed the replacement in certain areas of the white matter by masses of small nuclei, large compound granular corpuscles from which fat had been dissolved and large neuroglial cells; the myelin sheaths could not be recognized in these areas. There was no evidence of inflammatory change. The areas in which the characteristic appearances of Schilder's disease were observed, were symmetrically arranged on either side of the brain. The lesions did not appear to have advanced as far as those described in most of the records of the occurrence of the disease in mankind. The author compared the appearances of the lesions with those of a human specimen in his possession and concluded that there could be no question as to the identity of the condition observed in the monkey with Schilder's disease. No abnormality was found in any other organ with the exception of the liver, where there were some few areas of lymphocytic infiltration round the branches of the portal system. The author points out that while the monkey is a useful susceptible animal for experimental purposes the possibility of mistaking the natural for the experimental disease should not be overlooked when attempts are made to transfer the disease from mankind to the monkey.

Mixed Tumours of the Salivary Glands.

D. H. Patey (*The British Journal of Surgery*, October, 1930) reaches the conclusion that the mixed tumours of the salivary glands are a composite group of epithelial tumours, the varying pathological features of which depend on the degree of differentiation attained, the rapidity of division of cells, and the amount of myxomatous change undergone by the epithelium. According to the degree to which these changes have taken place, all gradations may be encountered between a myxoma on the one hand

and a highly differentiated tumour with ducts and acini on the other, and between a slowly growing tumour with regular cells and an irregular anaplastic growth.

Tumours of the Urachus.

R. C. BEGG (*The British Journal of Surgery*, January, 1931) has made an investigation of tumours of the urachus. He reports a case of his own and discusses forty-four from the literature. He discusses the anatomy of the urachus and shows that on theoretical grounds colloid adenocarcinomata resembling rectal growths may arise in the muscular wall of the upper part of the bladder. There is definite evidence, both *post mortem* and clinical, that such growths do occur as primary tumours and that the general tendency is for neoplasms in this region to assume the form of colloid cancer. Primitive cells of the urachus are always present in the muscular wall at the apex of the bladder. These are totipotent, but they tend to form tumours of the intestinal type. Adenomatous and cystic formations of various kinds are commonly found in connexion with the lower end of the urachus and the connexion between these and the colloid cancers can be followed through all the stages. All tumours of the bladder apex must be assumed to be of urachal origin from the point of view of treatment, unless the contrary be proved.

MORPHOLOGY.

Morphogenesis in the Anterior Portion of the Spinal Cord.

S. R. DETWILER (*Journal of Comparative Neurology*, August 15, 1930) continues his investigation into the effects produced by substituting one spinal cord segment for another in amblystoma embryos. Evidence has been accumulated which indicates that the anterior end of the embryonic spinal cord (particularly the first two segments) possesses an inherent capacity for extensive cellular proliferation as compared with more caudally lying segments and the caudal end of the *medulla oblongata*.

The Capillaries of Skeletal Muscles.

G. H. PAFF (*Anatomical Record*, September 25, 1930) records the results of counts of the numbers of capillaries per unit cross-sectional area of homologous muscles of the rat, guinea-pig and cat. In general, the muscles of the rat are more richly supplied with capillaries per unit cross-section area than are those of the guinea-pig; the muscles of the guinea-pig in turn are more generously provided with capillaries than are those of the cat. The number of capillaries in these animals varies from 2,341 to 3,574 per square millimetre cross-section of muscle and connective tissue. The ratio of muscle tissue to connective tissue is approximately constant in a cross-section, whatever the size of the muscle fibres. In tissue prepared by the paraffin technique this ratio is approximately seven to three. An inverse proportion exists between the predicated intensity of metabolism and the area of muscle tissue supplied by one capillary.

Total Glomerular Counts in Hemihypertrophy.

R. A. MOORE (*Anatomical Record*, September 25, 1930) enumerates the total glomeruli in each kidney of a person in whom the condition of so-called hemihypertrophy existed. The results contribute to the general thesis that the number of glomeruli in the kidney is independent of either acquired or congenital defects in the opposite kidney.

Splenectomy in the Salamander, Triturus Viridescens.

H. E. JORDAN AND C. C. SPEIDEL (*American Journal of Anatomy*, June 15, 1930) state that in the salamander, *Triturus viridescens*, the spleen is the chief organ for erythrocytopoiesis and thrombocytopoiesis, the liver capsule, for granulocytopoiesis. After total splenectomy new erythrocytes are differentiated chiefly in the general circulation, to some extent in the heart and in the sinuses of the liver. Proliferation by mitosis of young red cells and of their hæmoblast progenitors occurs in the general circulation. Of special interest is the failure of the granulocytopoietic capsule of the liver to act as a compensatory locus of erythrocytopoiesis. There is probably an increased rate of production of lymphoid hæmoblasts from the reticular cells and lymphoid cells of the liver capsule. These lymphoid hæmoblasts subsequently migrate into the sinuses of the liver and the general circulation. Thrombocytes in all stages of development were observed in the blood of some of the salamanders kept for one year after splenectomy. The thromboblasts revealed their origin from the lymphoid hæmoblast (hæmocytoblast). The authors state that this study emphasizes the importance of the general circulation in the salamander as a potential locus for both erythrocytopoiesis and thrombocytopoiesis. In no case was there formation of a new spleen, although sixty of the animals were kept for four months to one year after total extirpation of the spleen.

The Macrophages of Living Amphibian Larvæ.

E. R. CLARK AND E. L. CLARK (*American Journal of Anatomy*, July 15, 1930) give an account of the life history, the chief morphological characteristics, and many of the reactive powers of the tissue macrophages obtained by observation of the living cells in the transparent tail fins of amphibian larvæ. Individual cells were observed uninterruptedly during periods of four to thirty-six hours' duration. The authors state that it is clear that the macrophage is a very early cell to differentiate. It develops in the tissue of the tail from an undifferentiated strand of primitive mesoderm at the same time as do other varieties of early connective tissue cells and entirely independently of vascular endothelium or of any kind of blood cell. It has been observed to increase by mitotic division, but has never been seen to change permanently into any other forms of blood or tissue cell. It is the hardiest and most resistant cell in the region studied. It always displayed increased activity in the presence of injuries to the surrounding tissue. Individual cells marked with carmine were watched for as long as three weeks, during which period they showed not the slightest sign of degeneration.

Monocytes of the Blood and Tissue Macrophages.

E. R. CLARK AND E. L. CLARK (*American Journal of Anatomy*, July 15, 1930) state that it seems certain from their observations that the monocytes of the blood in amphibian larvæ are identical with the clear mononuclear phagocytes of the tissues and that they are both capable of enlarging to form typical tissue macrophages. The large pigmented macrophage is therefore a monocyte, which has emigrated from the blood vessel and has been carrying a phagocytosis. Regarding the source the monocytes of the blood stream, their observations supply no data whatever.

The Intervertebral Discs.

N. ROSS SMITH (*The British Journal of Surgery*, January, 1931) has investigated the structure of the intervertebral discs, their mode of nutrition and their state from infancy to old age. The material investigated was obtained from the bodies of fifty-seven persons, whose ages varied from one to eighty-six years. The author concludes that the discs are rudimentary diarthrodial joints possessing a cavity filled with villi, a fibrocartilaginous capsule, and upper and lower plates comparable to articular cartilages. He points out that the cartilaginous plates are properly parts of the disc and not of the vertebræ, as usually described. The discs are nourished by blood channels which pierce the cartilaginous plates from the marrow of the vertebral bodies. In the first two decades of life an intervertebral disc is biconvex and highly elastic. The *annulus fibrosus* consists of obliquely running parallel laminæ of white fibrocartilage with a few superficial layers of ordinary fibrous tissue. In the first two decades of life the fibres of the annulus are fine and white, the cartilaginous plates are thin, translucent and bluish-white, and the nucleus is amorphous, gelatinous, colourless and sharply defined. The nucleus varies in size and position relative to the annulus. In subsequent decades the disc tends to become progressively more coarse, inelastic, tough and discoloured. The author found that changes were as common and severe in the thoracic as in the lumbar discs, but discs in the two regions were not always affected simultaneously or to the same degree. In some individuals the discs preserve their juvenile state until late in life.

Special Articles on Diagnosis.

(Contributed by Request.)

XLI.

DIABETES MELLITUS.

Recent advances in knowledge have so completely altered our view point that, while ten years ago glycosuria and *diabetes mellitus* were almost synonymous terms, today the discovery of sugar in the urine merely indicates the imperative necessity of carrying out further tests to explain its presence.

Sugar is still, however, quite often found in the urine for the first time during a routine examination such as that carried out for insurance purposes, and unsuspected diabetes is still a common cause for a supposedly healthy person being suddenly at death's door, though, happily, the door is now usually slammed by insulin.

It will be well, therefore, first to review briefly the type of symptoms that should suggest this diagnosis as a possibility, bearing in mind that, under certain conditions, such as those obtaining in contract practice, the routine examination of the urine of every patient presenting himself for treatment is scarcely practicable.

Abnormal thirst and polyuria are almost invariable in advanced cases, but neither may be complained of in an early stage of the disease. Loss of weight despite a normal or voracious appetite is very suggestive. Insidious pulmonary tuberculosis with disproportionate wasting should remind us that tuberculous lesions often light up in a mysterious way in diabetes.

Chronic gall bladder disease often leads to chronic pancreatitis, and when a patient who has refused surgical treatment for his gall stones, begins to waste, he has usually developed either malignant disease or diabetes. A test of the urine will sometimes explain a neuritis or a neuralgia, especially a symmetrical neuralgia such as a bilateral sciatica. It is well to carry out a similar test before referring a patient with failing eyesight to a specialist.

So many skin lesions are associated with diabetes that it is a good plan to look for sugar when confronted with any of them. Pruritus and eczema of the genitals are, perhaps, the most common, but boils and carbuncles are also to be thought of as the special prerogative of the diabetic. It must not be forgotten, however, especially when giving a prognosis, that occasionally the sequence is the other way, and that any acute suppurative lesion may occasionally set up an acute type of diabetes that will clear up with the primary lesion. Gangrene in diabetes occurs only in elderly patients and really results from coincident vascular disease, but its appearance indicates a prompt examination of the urine. *Xanthoma diabeticorum* is quite a rarity, but its resemblance to other varieties of xanthoma suggests the exclusion of glycosuria in the case of anyone presenting this type of lesion.

Certain patients with disease of other endocrine glands are prone to have sugar in the urine, especially those with acromegaly and hyperthyreoidism.

When it comes to testing the urine, it is wise to remember that, if the condition is mild, sugar may be absent from urine obtained in a fasting stage. The early morning sample so favoured by the laity is by no means the best for the first, crucial, test. The ideal specimen is one passed about an hour after an ordinary meal. Of the many known tests for the detection of sugar in the urine, that with Fehling's solution is still probably the most popular, but of recent years Benedict's test has taken its place to a considerable extent. Though the latter test is regarded by the American school as more foolproof, actually nearly every substance that produces partial reduction of Fehling's gives also slight reduction with Benedict's test. Moreover, in this test considerable care has to be exercised in measuring exactly eight drops of urine with a dropper. If the copper and alkaline components of the Fehling's solution are kept in separate bottles and mixed only just before boiling for the test, no one with reasonable common sense should go astray.

Though there are certain fallacies in the test, it is comforting to remember that it will never fail to detect sugar when present. No reduction means no sugar. On the other hand, an immediate and complete reduction, with reddish or yellow deposit, when equal quantities of the solution and the urine are boiled together, always indicates the presence of sugar. Difficulty arises when there is a dirty greenish or greenish-yellow opalescence with or without a certain amount of similar coloured muddy-like deposit, for there are several substances other than a small amount of sugar that may produce such a reaction.

Albumin in any quantity will, of course, be thrown down by boiling, and this will render the solution cloudy. Albumin should, therefore, always be looked for before sugar and, if present, be precipitated and filtered off before proceeding with the test for sugar.

The most common and innocent partial reductions occur when creatinine, uric acid, xanthin bodies or urates are present in abnormal quantities. They should be suspected if the urine is highly concentrated and of high specific gravity, as diabetic urines of high gravity are usually pale and may be expected to give prompt and complete reductions. If the test is repeated after diluting the urine with an equal quantity of water, it should give no reaction in the absence of glucose. Or these substances may be precipitated by adding five cubic centimetres of a 10% solution of sodium acetate and the filtrate tested for sugar. Any doubt in this type of case may be set at rest by telling the patient to abstain from protein food for a day or two and drink plenty of water and then supply a specimen of urine passed an hour after a meal rich in carbohydrates. If the first reaction was due to glucose there will be no question about the second.

Glycuronic acid, an intermediary product in carbohydrate metabolism, reduces the copper solution and may appear in considerable quantities in the urine of those taking chloral hydrate, chloroform, camphor, benzoic acid, phenol and certain other aromatic drugs. The obvious course in such cases is to test again when the patient is no longer taking the drugs.

With these exceptions a reduction of Fehling's (or Benedict's) solution is due to sugar. Usually this is glucose (dextrose), but certain other sugars will produce similar reaction and are occasionally present in the urine. The only ones of practical interest are lactose (milk sugar), lævulose (fruit sugar) and pentose. Lactose should be suspected in the case of a pregnant or nursing woman. That it is not glucose is shown by its not fermenting when active yeast is added to it in a ureometer. Lævulose is practically never found alone, but only in the urine of some diabetics along with glucose. It can be identified only by rather elaborate tests and it ferments with yeast. As, however, a blood sugar estimation will show that diabetes is present, the differentiation of lævulose is only of academic interest. Pentose is a form of sugar which appears to be useless to the body, and so, when present, it is continuously excreted in the urine. It is especially found in the urine of Jews. It usually only changes the Fehling's solution after prolonged boiling, but then the reaction is sudden and complete. It does not ferment with yeast, and it can be identified by certain special tests, but a sugar tolerance test will show that the patient has not got diabetes.

The urinary tests for acidosis should automatically follow upon the finding of sugar in the urine. If the results are positive, they indicate not only that diabetes is present, but that it is in a stage calling for prompt remedial measures. In the absence of acidosis the completion of the diagnosis by examination of the blood sugar can proceed on more leisurely lines. Clinically the presence of acidosis is recognized in the urine by testing for the presence of acetone and diacetic acid.

Simplest and most generally useful is the ferric chloride test for diacetic acid, but it should be remembered that a dense precipitate of ferric phosphate often forms at first, especially abundant in phosphatic urine, but that it dissolves on further addition of the ferric chloride. In the

absence of diacetic acid the solution remains yellow, but in its presence some shade of Burgundy red replaces the yellow. As a precisely similar colour appears if the patient has taken some aspirin or other salicylic product, antipyrine, cyanates, or acetates, the solution should always be boiled for a couple of minutes at least. If the colour remains, it is not due to diacetic acid, which, on boiling, changes to acetone and promptly evaporates, leaving the solution of the original yellow colour.

With no reaction to a diacetic acid test there is no cause for anxiety. Rothera's nitroprusside test for acetone is more delicate, and if it is obtained when the ferric chloride test gives no reaction, the amount of acetone is so small as to be negligible from an immediate prognostic point of view.

The actual testing of the blood sugar, though not difficult, demands the devotion of more time than can usually be spared by those in active practice and it is therefore usual to refer the patient to a consulting pathologist or biochemist for the investigation. In order to appreciate the significance of reports from these sources, it is necessary to have a working knowledge of present day views in regard to carbohydrate metabolism.

Carbohydrate in the bodily tissues occurs in two forms, glucose, which might be described in banking terms as a liquid asset, and glycogen, a fixed deposit. Glycogen is an intermediate product into which glucose is readily converted, and which is apparently equally easily converted back into glucose. Under normal conditions glucose is maintained in the blood, except just after a meal, at a very constant level. The other tissues of the body are all apparently capable of holding a considerable, but so far unmeasured, quantity of glucose as such, but normally any amounts of glucose above a certain quantity are rapidly converted into glycogen, which is stored away, especially in the liver and muscles, to be subsequently utilized to keep up the level of glucose in the blood when food is not being taken. Glucose can be combusted and utilized by the tissues only in the presence of insulin, an internal secretion of the pancreas, and the amount of his glucose that is available to any person as a source of energy, depends upon his capacity to secrete sufficient insulin for its combustion. A normal pancreas, however, can secrete far more insulin than is required to deal with the ordinary requirements of the body. Insulin also apparently plays a not yet fully understood part in glycogen metabolism.

Once it was established that the fasting blood of normals contained a constant amount of glucose and, further, that the increase in this amount that followed the ingestion of a known quantity of carbohydrate was also constant, it was obvious that examination of the blood sugar would be a very great help in the diagnosis of diabetes and other disturbances of carbohydrate metabolism. Normal fasting blood contains very constantly about 100 milligrammes of glucose per 100 cubic centimetres and variations below 80 milligrammes and above 120 milligrammes may be accepted as indicating a disturbed carbohydrate metabolism. This is most usually recorded as a percentage and the normal level put down as between 0·08% and 0·12%. After the ingestion of food leading to the absorption of glucose there is a rise in the blood sugar level which varies with the amount absorbed, but even with quite gross amounts does not rise above 0·16%. The maximum level, usually spoken of as the peak, is reached within the first hour and then there is a rapid fall, and within two hours the level is generally slightly below the starting point.

Under certain conditions of abnormality the blood sugar rises above the peak level of 0·16%, and this is spoken of as hyperglycæmia. Theoretically, such a high level may be due to: (i) Great excess of ingested carbohydrates, (ii) defect in the storage apparatus—the liver or muscles having some defect that prevents them storing glycogen quickly, (iii) deficiency of insulin. Practically, the efficiency of the pancreas is so great that (i) the great excess of ingested carbohydrate is negligible as a cause of hyperglycæmia in normal people.

When the blood sugar level rises appreciably above 0·16%, a safety valve action of the kidneys comes into play and they secrete the surplus sugar as fast as they can. It may be noted in passing that the old idea that the sugar in the urine was the disease led to the name of *diabetes mellitus*.

The exact level at which the kidney action comes into play, often spoken of as the leak point, varies, but it is usually at about 0·18%, tending to become higher in long standing diabetes. In quite a number of otherwise normal people, however, the leak point is low, usually somewhere between 0·12% and 0·16%. Such people tend to secrete sugar after a normal meal, but their blood sugar does not rise above normal. The condition is known as renal glycosuria and is apparently harmless and not liable to develop into diabetes, though it does occasionally happen that a true diabetic has also a low leak point.

It is clear, then, that to decide the true nature of a glycosuria it is indispensable to investigate the blood sugar. Such an investigation may take one of two forms: (i) A single estimation of the fasting blood sugar. (ii) A series of estimations at intervals after the ingestion of carbohydrates. This latter is known as a glucose tolerance test and the graph on which the figures obtained are usually plotted out is spoken of as a glucose tolerance curve.

In many cases the single estimation shows the level of the fasting blood sugar to be so high that a diagnosis of *diabetes mellitus* is immediately established so that a sugar tolerance test is unnecessary for this purpose. Indeed, in dealing with patients who have a very high blood sugar concentration, particularly with diacetic acid in the urine, such a test, at least at this stage, is highly undesirable.

It is especially when the fasting blood sugar is either normal or only a little abnormal that a tolerance test is an essential part of the diagnosis. Various types of tolerance tests are in vogue, but the most usual one involves the determination of the blood and urinary sugar immediately before, thirty minutes, one hour, one hour and a half, and two hours after the ingestion of 50 grammes of glucose in 150 cubic centimetres of water. The figures obtained from the blood sugar estimation are plotted on a graph already spoken of as a glucose tolerance curve.

Five main types of curve have been recognized:

1. The normal curve with figures within the limits of those already spoken of as characteristic of normality.

2. Normal fasting blood sugar, normal peak, but a slower return to normal than usual—a lag curve as it is sometimes called. This is not due to diabetes, but to an increased tolerance for carbohydrates, and is met with in certain endocrine deficiencies, *arthritis deformans* and certain other conditions.

3. Normal fasting level, high peak, normal return. This is not diabetes, but characteristic of defective storage. It is seen in liver defects, as after "Salvarsan" poisoning, and in cirrhosis of the liver.

4. Normal fasting level, high peak, slow return.

5. Abnormal curve throughout—high fasting level, high peak—often reached after the first hour, slow return.

Only the last curve justifies a definite and immediate diagnosis of *diabetes mellitus*.

Curve 4 may mean that diabetes is present, especially when it is met with in a patient who has been carefully dieted before the test. When this doubt arises the question can be settled by repeating the test after the patient has been allowed a full ordinary diet for a day or two to insure normal bodily stores of glycogen. If diabetes is present, the curve obtained will be of the 5 type.

Additional information is gathered from the examination of the specimens of urine passed during the test.

If sugar is found in some samples with Curves 1 and 2, the case is one of renal glycosuria, but if it is present in all the specimens, it is probably pentose, and, if so, will not ferment with yeast. It can be identified then with certain special tests, but neither renal glycosuria nor pentosuria is of serious significance.

In Curves 3, 4 and 5 glucose is to be expected when the peak reaches 0·18%, though in some diabetics it may not appear till a higher level is reached. Sugar appearing at lower levels has the same significance as in 1 and 2.

When by one or other of the above tests the presence of *diabetes mellitus* has been established it should be

borne in mind that the disturbance of pancreatic function may not be in any given case a complete diagnosis. An exhaustive examination of the general health should always be made in a diligent search for any other departure from normal. Timely discovery and treatment of gall bladder disease, pancreatic cyst or pancreatic calculus, may lead to complete disappearance of symptoms, as also may surgical treatment of hyperthyreoidism and acromegaly. It has already been indicated that the discovery of a recent pyogenic infection gives reason for hope that its cure may be followed by recovery of the pancreas.

Other conditions, as for example, chronic nephritis and pulmonary tuberculosis, though they may not be ætiological factors, will need to be taken into consideration in planning treatment and serve as a reminder that in this, as in all disease, the treatment must be individual.

C. Bickerton Blackburn, M.D. (Sydney),
Honorary Physician, Royal Prince Alfred Hospital, Sydney.

British Medical Association News.

MEETING OF THE FEDERAL COMMITTEE.

A meeting of the Federal Committee of the British Medical Association in Australia was held at the Medical Society Hall, East Melbourne, on March 27, 1931, Sir Henry Newland, C.B.E., D.S.O., the Chairman, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. R. H. Todd, Dr. T. W. Lipscomb.
Queensland: Dr. D. G. Croll, C.B.E., Dr. W. N. Robertson, C.B.E.
South Australia: Sir Henry Newland, C.B.E., D.S.O., Dr. Bronte Smeaton.
Tasmania: Dr. Gregory Sprott, Dr. A. W. Shugg.
Victoria: Dr. F. L. Davies, Dr. J. Newman Morris.

Sir Henry Newland and Dr. R. H. Todd acted as proxies for Dr. F. A. Hadley and Dr. D. D. Paton, of the Western Australian Branch, respectively.

Minutes.

The minutes of the previous meeting of October 2, 1930, copies of which had been sent to the members after the meeting, were submitted and signed as correct.

Financial Statement.

The financial statement for the six months ended December 31, 1930, prepared by Messrs. Coates, Cunningham and Company, Chartered Accountants, and audited by Dr. W. H. Crago, the Honorary Auditor, together with the Australasian Medical Congress (British Medical Association) accumulated funds account, was presented and adopted.

It was decided, on the motion of Dr. Gregory Sprott, seconded by Dr. A. W. Shugg, to suspend the contribution altogether for the current year.

Appointment of Office Bearers.

Sir Henry Newland was appointed Chairman and Dr. J. Newman Morris Vice-Chairman of the Federal Committee. Dr. R. H. Todd was appointed Honorary Secretary and Honorary Treasurer. Dr. Todd was granted seven months' leave of absence and the arrangements for a substitute to act as Honorary Secretary and Honorary Treasurer were left in the hands of the Chairman.

Dr. W. H. Crago was appointed Honorary Auditor.

British Medical Association Scholarships and Grants.

It was reported that application had been received from a member for a British Medical Association scholarship. The application would be forwarded to the headquarters of the Association as soon as the supporting statements from referees had been received.

Subscription from Overseas Branches.

The question of a reduction of the annual subscription paid by the Branches in Australia on account of members from £1 11s. 6d. to £1 7s., which had been the subject of a communication with the Central Council, was again discussed.

It was decided that Dr. R. H. Todd should be empowered to discuss the matter with the Medical Secretary in London.

Federal Council.

Further consideration was given to the proposals for the formation of a Federal Council representative of the Branches in Australia. The draft Memorandum and Articles of Association and By-Laws were published in The Medical Journal of Australia of January 18, 1930, at page 92. At the previous meeting discussion had centred around Article 14. This article read:

> The number of members of the Executive Committee inclusive of *ex officio* members shall not be less than three nor more than six.

The New South Wales Branch had suggested, as it had done on a previous occasion, that in Article 14, (i) the word "not" be omitted, also the words "less than three nor more than"; and words be added to the effect that "no more than one member representing any one Branch shall be a member of the Executive Committee" so as to make the article read: "The number of members of the Executive Committee, inclusive of *ex officio* members, shall be six; and no more than one member representing any one Branch shall be a member of the Executive Committee." It was resolved at the previous meeting that the first part of the suggested alteration of the New South Wales Branch be adopted, so that the article would read: "The number of members of the Executive Committee, inclusive of *ex officio* members, shall be six." The Honorary Secretary reported that all the Branches with the exception of the New South Wales Branch had approved of this alteration.

The matter was reopened for discussion.

Dr. D. G. Croll said that the Queensland Branch had always asked for a small committee. He did not think that an executive of six would give the continuity that was wanted. He referred to the appointment of a permanent secretary as desirable and said that there should be someone to coordinate the work of the Council.

Dr. F. L. Davies said that committees could be appointed for specific purposes, but they were no use in emergencies. There would in the future be many matters that would have to be decided on very short notice.

Dr. T. W. Lipscomb said that if an occasion such as that mentioned by Dr. Davies arose, an executive committee would have no time to act. The Chairman would have to act. He was in favour of an executive with a quorum of two.

Dr. J. Newman Morris agreed with Dr. Lipscomb and said that they all wanted workability and power to act.

Dr. R. H. Todd said that if provision were made for substitutes and proxies in the Executive Committee the difficulties would be overcome. He suggested by way of amendment the addition to Clause 14 of the words: "Not more than one member representing any one Branch shall be a member of the Executive Committee" and the addition of a new clause as follows:

> Article 23 (*a*).—"When any member of the Executive Committee shall be unable to attend any meeting thereof, the Council of the Branch which such member shall represent shall be entitled to nominate another member of the Federal Council, either the other member thereof representing such Branch or a member representing another Branch, whether such member be a member of the Executive Committee or not, to act as a substitute for the member who shall be unable to attend, and, if it shall not nominate any such substitute, the member who shall be unable to attend shall himself be entitled to appoint another member of the Executive Committee to be his proxy for such meeting.

The appointment of a substitute or a proxy shall be in writing, and if made by the Branch Council shall be signed by the Secretary of the Branch and if made by the member shall be signed by him, provided that, in a matter of urgency, the appointment may be by telegram or telephone. A substitute appointed as aforesaid shall also be entitled to appoint in writing or in a matter of urgency by telegram or telephone, another member of the Executive Committee to be his proxy if he so desire. Any proxy may be limited to any part or session of a meeting of the Executive Committee or to any specified question or questions, and must be produced at the meeting at which the holder proposes to vote."

The amendment was carried unanimously. It was also decided that two members, representing one or more Branches, should be a quorum at any meeting of the Executive Committee. By-Law 4 was amended to read accordingly.

The Honorary Secretary reported that a letter had been received from the Medical Secretary of the British Medical Association in confirmation of a cablegram approving generally of the Draft Constitution of the Federal Council but suggesting amendments of Articles 12 and 45 and enclosing a "Draft Agreement prepared by Solicitors of Association between Federal Council and Association."

The suggestions of the Medical Secretary were approved.

It was decided:

That the proposal for the formation of a Federal Council representative of the Branches in Australia be submitted on behalf of such Branches to the Council of the British Medical Association in accordance with Article 16 and By-Law 23 of the British Medical Association, together with the proposed Memorandum and Articles of Association and By-Laws of the Federal Council as now drawn up and approved by the Federal Committee and the several Branches in Australia.

Model Rules Governing Procedure in Ethical Matters.

It was reported by the Honorary Secretary that the model rules governing procedure in ethical matters had been adopted by all the Branches.

Poliomyelitis.

A proposal had been received from the Victorian Branch that a request should be made to the Commonwealth Government for an annual grant towards the curative treatment of patients suffering from the effects of poliomyelitis. The matter had been placed before the Branches. The New South Wales Branch had approved of the suggestion as far as early treatment was concerned.

In supporting the proposal, Dr. Newman Morris referred to the difficulty of securing hospital accommodation for patients requiring treatment extending over a long period. The number of invalid pensions granted was enormous (600 had been allotted since 1925) and there was no doubt that with adequate treatment the number would be reduced. Added to this there was the belief that there was an epidemic ahead of them. Dr. Morris said that the Federal Treasurer had had an investigation made into the condition of invalid pensioners to ascertain how many could be made to earn their own living. The number had been only 1%. He moved that the proposal of the Victorian Branch be acted upon.

In seconding Dr. Morris's motion, Dr. A. W. Shugg pointed out that many of the persons affected by poliomyelitis in epidemics up to the year 1918 were in receipt of invalid pensions, but that none of those affected in the last epidemic had yet arrived at the stage of needing pensions. The Commonwealth Government was soon to be faced with applications for pensions from many of these people. Dr. Shugg referred to some patients treated by him in the southern part of Tasmania. Of 66 persons 15 had been transformed from becoming incipient receivers of pensions and were able to earn for themselves. These persons were all young and their expectation of life was estimated at forty-five years, so that the saving to the Government in the recovery of these persons due to the non-payment of pensions could be estimated at £35,000. Expenditure of a small sum annually was necessary and he was convinced that this should be devoted to both early and late treatment.

It was resolved that Dr. A. W. Shugg should be appointed to act as a subcommittee and to report to the Chairman.

Australian Inland Mission Frontier Medical Service.

Reference was again made to the question of the Australian Inland Mission Frontier Medical Service. A "Draft Prospectus of the Aerial Medical Services of Australia" had been received and had been sent to members of the Committee. In the absence of further definite information, the matter was allowed to stand over to the next meeting.

Australasian Medical Congress (British Medical Association).

A proposal received from the Executive of the fourth session of the Australasian Medical Congress (British Medical Association) that the subscription for the fourth session for attending members should be three guineas and for non-attending members two guineas was approved.

Approval was given to the proposal of the Executive of the fourth session that the following distinguished persons should be invited to become patrons of congress: The Chancellor of the University of Western Australia, the Lord Mayor of the City of Perth, the Premier of Western Australia. It was also decided that invitations to be patrons should be sent to the following: The Governor-General of the Commonwealth, the Governor-General of the Dominion of New Zealand, the Governor of New South Wales, the Governor of Queensland, the Governor of South Australia, the Governor of Tasmania, the Governor of Victoria, the Governor of Western Australia, the Governor of Fiji, the Lieutenant-Governor of Papua.

It was resolved that, in accordance with the regulations governing congress, the following persons be invited by the Federal Committee to be Vice-Presidents of Congress: Past Presidents of Congress, Vice-Presidents of the British Medical Association in Australasia, the Director of Naval Medical Services of the Commonwealth, the Director-General of Army Medical Services (Commonwealth), Director-General of Army Medical Services (New Zealand), Director-General of Health (Commonwealth), and the Director-General of Health (New Zealand).

It was resolved that the following words be added to Regulation 3 (*b*) (3): "Provided also that the Editor of THE MEDICAL JOURNAL OF AUSTRALIA be a member *ex officio* of the Executive Committee."

Repatriation Department.

A letter had been received from the South Australian Branch in regard to the War Pension Assessment Tribunals, asking the Federal Committee to consider: (i) Whether under the *Australian Soldiers' Repatriation Act*, 1920-9, and Amendment Number 74 of 1930, it is desirable that the medical members of the Assessment Appeal Tribunal should have no option but to recommend that the present pension continue in cases where the Tribunal considers the pension excessive or the pensioner entitled to no pension; (ii) whether it is desirable that the medical history of the appellant should be elicited by the Chairman of the Tribunal rather than by one of the medical members; and to take what action it deemed necessary under (i) and (ii).

After discussion it was resolved:

That Assessment Appeal Tribunals should have the option not only of increasing, but also of decreasing and discontinuing pensions and that a copy of the resolution be sent to the Federal Minister concerned.

Medical Services to Natives in Central Australia.

At the last meeting of the Federal Committee further consideration was given to the question of the provision of a medical service to the natives in Central Australia. A report of a subcommittee consisting of Dr. J. Newman Morris, Dr. F. L. Davies and Dr. G. Simpson was received

at that meeting and further action was deferred. The report had been in the hands of members for some little time and it was resolved that a copy of the report be sent to the Department of Internal Affairs and the Department of Health of the Commonwealth. It was also resolved that the report be sent to The Medical Journal of Australia for publication.[1]

Health Research Council.

Mention was made of the question of the establishment of a Health Research Council and of the reply received from the Minister of Health of the Commonwealth, recorded in the account of the last meeting. It was resolved to allow the matter to stand over.

Duty on Insulin.

At the previous meeting mention had been made of the question of the duty on insulin and it was decided to communicate with the Department with a view to the removal of the duty. The Minister, as reported on a previous occasion in this journal, had taken no action in the matter. It was the opinion of members of the Committee that the Minister in his reply to the Committee had missed the points at issue and that definite hardship was inflicted on sufferers with diabetes by reason of the duty. It was decided to make further representations to the Minister.

Registration in the United Kingdom of Australian Graduates.

A letter from the Registrar of the General Medical Council was read dealing with the registration of Australian graduates in the United Kingdom. It was pointed out that the General Medical Council cannot recognize the Commonwealth as a single entity, but that the States must still be regarded independently. It is necessary for an Australian practitioner desirous of registering in the United Kingdom to be registered in the State from the university of which he has obtained his qualification.

Date and Place of the Next Meeting.

It was determined that it be left to the Chairman to fix the date and place of the next meeting.

Thanks.

Votes of thanks were accorded to the Chairman, Sir Henry Newland, for presiding and to Dr. F. L. Davies and Dr. J. Newman Morris and the Council of the Victorian Branch for their hospitality and for the accommodation provided for the meeting.

NOMINATIONS AND ELECTIONS.

The undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Reed, Joseph Clynton, M.B., B.S., 1929 (Univ. Melbourne), Children's Hospital, Carlton, N.3.

Hospitals.

THE ROYAL PRINCE ALFRED HOSPITAL.

The Treatment of Syphilis.

At the first annual reunion of the Royal Prince Alfred Hospital Residents' and Ex-Residents' Association, held from October 7 to 11, 1930, the medical officers of the Department for the Treatment of Venereal Diseases made the following statement of routine methods of treatment adopted by them.

All infected persons showing a suspicious primary sore are subjected to the dark ground illumination test, on three consecutive days, if necessary. If the sore has been present three weeks or more, a Wassermann test and a Kahn flocculation test are done also, but in no instance is treatment commenced until a definitely positive diagnosis is established. Secondary and tertiary lesions are diagnosed by the Wassermann test *et cetera*. The first course of treatment usually given in the clinic is as follows:

First day, intravenous injection of "Novarsenobillon," 0·45 gramme.
Eighth day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Fifteenth day, intramuscular injection of "Muthanol," 2 cubic centimetres.
Twenty-second day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Twenty-ninth day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Thirty-sixth day, intramuscular injection of "Muthanol," 2 cubic centimetres.
Forty-third day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Fiftieth day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Fifty-seventh day, intramuscular injection of "Muthanol," 2 cubic centimetres.
Sixty-fourth day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Seventy-first day, intravenous injection of "Novarsenobillon," 0·6 gramme.
Seventy-eighth day, intramuscular injection of "Muthanol," 2 cubic centimetres.
Eighty-fifth day, intramuscular injection of "Muthanol," 2 cubic centimetres.
Ninety-second day, intramuscular injection of "Muthanol," 2 cubic centimetres.
Ninety-ninth day, intramuscular injection of "Muthanol," 2 cubic centimetres.
One hundred and sixth day, intramuscular injection of "Muthanol," 2 cubic centimetres.

Then follows one month's rest from treatment with potassium iodide 1·0 gramme (fifteen grains) and *liquor hydrargyri perchloridi* 2·0 mils (thirty minims) thrice daily for the first fortnight. At the end of the rest period a provocative injection of 0·45 gramme of "Novarsenobillon" is given and the Wassermann test carried out. The previous course of treatment is then repeated, whether the Wassermann test fails to yield a reaction or still gives a positive reaction. Another month's rest is given with iodides for the first fortnight and the Wassermann test is again carried out after a provocative injection. If the Wassermann test now fails to yield a reaction a course of ten to twelve weekly intramuscular injections of bismuth alone is given, followed next by six injections of "Novarsenobillon" and eight of bismuth. If the Wassermann test still fails to yield a reaction, as it should do, the treatment is continued with bismuth only till the patient has failed to react to the Wassermann test for two years.

At the end of that time the patient's blood is tested at intervals over a period of another two years. If, on the other hand, the Wassermann test still gives a positive reaction after the first or second courses and no intolerance to arsenic is exhibited, a further mixed course should be given, but with less arsenic, depending on the patient's tolerance. When it is difficult to produce failure of response to the Wassermann test, intramuscular injections of arsenic, for example, with "Sulpharsenol" or "Myosalvarsan" are tried, also "Quinby" intramuscularly or "Tryparsamide" intravenously. Alternation and change of drugs frequently produce improvement in the blood, but each change must be judged on the conditions present and the general health of the patient. When the Wassermann test yields a persistently positive reaction, not less than four years' treatment is given, and then one course every year to keep any symptoms from manifesting themselves. All intravenous injections are mixed in 10% sodium thiosulphate. Before injection all patients have their urine tested and are given an ounce of the following mixture to drink with an equal quantity of water, namely, glucose 56·0 grammes (14 drachms), sodium bicarbonate 1·0 gramme (15 grains),

[1] This report will be published in a subsequent issue.

oil of lemon 0·06 mil (one minim), water to 90·0 cubic centimetres (three fluid ounces).

This routine prevents as far as possible any toxic symptoms following administration of the drugs. An attempt is now being made to ascertain the response of the cerebro-spinal fluid to the Wassermann test with every patient once a year. Each patient is weighed at the beginning and end of each course and examined clinically once a year for signs of any possible nervous involvement.

Obituary.

JOHN MORGAN.

We regret to announce the death of Dr. John Morgan, which occurred on April 5, 1931, at Hunter's Hill, New South Wales.

Correspondence.

AN ASSOCIATION OF PHYSICIANS.

Sir: In your leading article having reference to the proposed formation of an association of physicians, with the further view of forming a Royal college, you refer to the clumsy title to which "including New Zealand" is appended.

I have been many times in New Zealand and have travelled with thousands of New Zealanders. The one thing we Australians must realize is that the people of New Zealand as a whole have a strong and increasing objection to the name of their country being submerged by such a bastard term as Australasia. They desire that full value be given to "New Zealand."

Why not say "The Association of Physicians of Australia and New Zealand"? Personally, I object to the word Australasia. It means Southern Asia. Both Australia and New Zealand should get full value for their geographical names.

Yours, etc.,

T. J. Henry, F.R.C.S. (Edin.).

Manly,
New South Wales,
March 30, 1931.

TONSILLECTOMY.

Sir: Much has been written in the correspondence on the method of performing the operation of tonsillectomy, and the practitioner will be beginning to regard this as a question that cannot be settled between the specialists themselves. Without a clear statement is forthcoming and one which is agreed upon by the majority of specialists, the practitioner may come to regard the operation of tonsillectomy in the same light as he views the drug to be used for a certain purpose in his practice, when wanting a cure for a certain complaint he finds that anything up to perhaps a dozen drugs are recommended. He can usually then decide that none of them is of any real value. If he finds that one drug, such as digitalis, is recommended for cardiac failure, or sodium salicylate for acute rheumatism, he will usually decide that it is a specific and well worth using. If, therefore, he can be convinced that the majority of specialists throughout the world are of a fairly settled opinion, he may then decide that the operation is, after all, successful when well performed by those capable of doing it.

I have recently returned from a tour of two years on the other side of the world and the thing that was more or less universal practice in the nose and throat clinics was their methods of tonsillectomy. In the Hajek Clinic, Vienna, children under thirteen were guillotined under general anæsthesia and those over, dissected under local. In the Rudolph Virchow in Berlin the same held good. In the Zent Rokus in Budapest, the dividing line was set at about twelve. In the London Hospital general anæsthesia was used for both children and adults, and those under eleven to twelve were guillotined and those older dissected. In The Kent County Ophthalmic and Aural Hospital the dividing age was a little lower. In Edinburgh under twelve to fourteen guillotined under general anæsthesia, and the adults dissected under local. Again, in both Los Angeles and San Francisco the same held good.

Surely, therefore, we can say that the majority of the world's specialists consider a guillotine operation well performed is quite satisfactory in children under ten to fourteen years of age, and that for those older patients, because they have usually had some attacks of tonsillitis or quinsy *et cetera* and have usually got adhesions which often require considerable force to break down if guillotined, a dissection operation is the method of choice.

In my opinion it is not so much the guillotine that should be condemned, but the speed with which the operation can be and often is performed. A light anæsthesia with ethyl chloride of thirty seconds' duration is ample for the removal of both tonsils and the adenoids and an inspection afterwards when all goes well, and this is the duration of anæsthetic often demanded by the surgeon. If he "misses," he then has very little time to go back before his patient is out of the anæsthetic. This is the real cause of tonsillar tissue being left behind by an expert with the guillotine.

If the anæsthesia were more prolonged and the surgeon would swab the throat and examine for remains and be prepared to go in after them, then an experienced person would rarely leave behind anything which should be removed. In other words, he can get it with the guillotine if he has the time and knows how to use it. But, as in older children and adults he will often have to go back, if careful, he prefers a long anæsthetic. If, then, he is going to have the long anæsthetic, he may as well dissect from the beginning, taking his time and attending to hæmostasis afterwards.

Yours, etc.,

Hugh G. Mitchell.

61, Collins Street,
Melbourne,
March 20, 1931.

Sir: Dr. Graham Brown gave an excellent *résumé* of the question. Forty years ago I began with the old guillotine method. Then, influenced by Ballenger, I took up dissection. In 1913 I adopted the reverse guillotine, but returned to dissection in 1916, except for hospital work. I believe after nearly five years' experience that it is almost criminal to deal with an adult's tonsil by any other method than by diathermy, and I think I am as expert as most with the reverse guillotine or dissection. An important point is that removal of tonsils is the province of the many. I doubt if 5% of private cases are sent to the specialist. It looks so easy and it really is so difficult. I deplore the lack of instruction in the matter of tonsils. A student would be ploughed if he did not know how to operate on an appendix or a hernia, yet in most cases his examiner could not remove a tonsil decently. To completely remove a tonsil with diathermy is not easy, but it is not more difficult than by any other method. I hold that it is not necessary to remove a tonsil completely, excepting in those patients who have had a peritonsillitis. Diathermy may be tedious, but if we consider the patient first, that is not a valid objection. Children undergo diathermy quite well and its effect upon their tonsils is much greater. The septic lingual tonsil and lower pole of faucial tonsil is more easily dealt with by diathermy than by any other method. It would be better if only experts removed tonsils. Since that can never be, students should be instructed. The more knowledge they obtain, the less likely will the recent graduate be to tackle a difficult case.

Diathermy is much cheaper, much safer and involves less loss of time, and is the only method that will give a perfect anatomical result in every case.

Yours, etc.,

W. Kent Hughes.

22, Collins Street,
Melbourne,
March 22, 1931.

Books Received.

CANCER, ITS ORIGIN, ITS DEVELOPMENT AND ITS SELF-PERPETUATION; THE THERAPY OF OPERABLE AND INOPERABLE CANCER IN THE LIGHT OF A SYSTEMIC CONCEPTION OF MALIGNANCY; A RESEARCH, by W. Meyer, M.D.; 1931. New York: Paul B. Hoeber. Royal 8vo., pp. 460, with portraits. Price: $7.50 net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, Edited by E. B. Krumbhaar, M.D. I, The Beginnings: Egypt and Assyria, by W. R. Dawson, F.R.S.E.; 1930. New York: Paul B. Hoeber. Crown 8vo., pp. 95. Price: $1.50 net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, Edited by E. B. Krumbhaar, M.D. II, Medicine in the British Isles, by Sir D'Arcy Power, K.B.E., F.R.C.S.E.; 1930. New York: Paul B. Hoeber. Crown 8vo., pp. 93. Price: $1.50 net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, Edited by E. B. Krumbhaar, M.D. III, Anatomy, by G. W. Corner, M.D.; 1930. New York: Paul B. Hoeber. Crown 8vo., pp. 99, with illustrations. Price: $1.50 net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, Edited by E. B. Krumbhaar, M.D. IV, Internal Medicine, by Sir H. Rolleston, Bart.; 1930. New York: Paul B. Hoeber. Crown 8vo., pp. 101. Price: $1.50 net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, Edited by E. B. Krumbhaar, M.D. V, Physiology, by J. F. Fulton, M.D.; 1930. New York: Paul B. Hoeber. Crown 8vo., pp. 156, with illustrations. Price: $1.50 net.

OPERATIVE OBSTETRICS ON THE MANIKIN FOR STUDENTS AND PRACTITIONERS, by C. B. Reed, M.D., F.A.C.S.; Philadelphia: P. Blakiston's Son and Company. Royal 8vo., pp. 330, with 254 illustrations. Price: $4.00 net.

DIE REGULIERUNG DER ATMUNG, GLEICHZEITIG EIN BEITRAG ZUR PHYSIOLOGIE DES VEGETATIVEN NERVENSYSTEMS, by W. R. Hess; 1931. Leipzig: Georg Thieme. Royal 8vo., pp. 137. Price: M. 10.50 net.

THE PATHOLOGY OF DIABETES MELLITUS, by Shields Warren, M.D., with a Foreword by E. P. Joselin, M.D.; 1930. London: Baillière, Tindall and Cox. Royal 8vo., pp. 223, with 83 engravings and two coloured plates. Price: 35s. net.

A MANUAL OF TUBERCULOSIS FOR NURSES, by E. Ashworth Underwood, M.A., B.Sc., M.B., Ch.B., D.P.H., with an introduction by J. R. Currie, M.A., M.D., M.R.C.P., D.P.H.; 1931. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 279, with illustrations. Price: 6s. 6d. net.

PRACTICAL ANÆSTHETICS FOR THE STUDENT AND GENERAL PRACTITIONER, by C. F. Hadfield, M.B.E., M.A., M.D.; Second Edition; 1931. London: Baillière, Tindall and Cox. Post 8vo., pp. 350. Price: 7s. 6d. net.

Diary for the Month.

APR. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 22.—Victorian Branch, B.M.A.: Council.
APR. 24.—Queensland Branch, B.M.A.: Council.
APR. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 30.—South Australian Branch, B.M.A.: Branch.
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MAY 6.—Victorian Branch, B.M.A.: Branch.
MAY 7.—South Australian Branch, B.M.A.: Council.
MAY 8.—Queensland Branch, B.M.A.: Council.
MAY 12.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 14.—Victorian Branch, B.M.A.: Council.
MAY 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 22.—Queensland Branch, B.M.A.: Council.
MAY 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 27.—Victorian Branch, B.M.A.: Council.
MAY 28.—South Australian Branch, B.M.A.: Branch.
MAY 28.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

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